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BUREAU OF HYGIENE AND TROPICAL DISEASES.

TROPICAL DISEASES BULLETIN.

Vol. 44.] 1947. [No. 12.

RABIES.

SORIANO LLERAS, A. Rabia en Colombia. [**Rabies in Colombia.**] *Repertorio Med. y Cirug.* Bogotá. 1947, July, v. 2, No. 5, 387–402.

The author sketches the history of rabies in Colombia from the early years of the nineteenth century when, in 1810, owing to the frequent occurrence of the disease, an Order was promulgated that all dogs in the Department of Antioquia were to be hanged, except those employed in the fields, which were to be kept tied up. This does not seem to have been obeyed, for another Order in 1813 gave the police right of entry to houses to look for dogs and the owners of any such dogs discovered were fined ; if the dog was rabid, the owner had to pay damages for anyone injured and was liable to 3 years' imprisonment, or hard labour, or 5 years' transportation. Nearly a century later, in 1907–8, rabies was observed in dogs, foxes, cows and horses, and it was the custom of the people to bury alive, leaving the head above ground, any animal showing symptoms and the doctor had difficulty in preventing the relatives of a human patient adopting the same method in a case where symptoms appeared four months after the bite. Several records are quoted of subsequent years up to 1940, and for the six years 1940–45 more details are given in the form of protocols. The " numbers of treatments " [? persons treated] have steadily increased, the figures being 4,630, 5,730, 8,166, 9,032, 10,170 and 10,897. All patients are given a card on which are stated the site bitten, the characters of the wound and the results of treatment ; this card is suppposed to be returned two months after treatment is stopped, but less than 25 per cent. have been returned. The numbers of fatal cases during the same six years were : 25, 26, 29, 17, 20 and 12. Pasteur's form of treatment was used at first, but since 1929 Semple's method has been adopted. The author ends with these words : " The summary made in the present article shows that of 125 human cases [fatal cases] 54 had received the complete course, 71 had had no treatment at all or only a very few injections. Of those who died in spite of full treatment some did not start till many days after infliction of the bite." [There is nothing to show how the figure 125 is arrived at. Those given for the period 1940–45 total 129 ; if we exclude 25 dying in 1940 of whom, it is stated, there is no record of how many received treatment and how many did not, the total, as shown in the tables, is 104.] *H. Harold Scott.*

REMLINGER, P. & BAILLY, J. Action du latex de figuier sur le virus de la rage. [**Action of the Latex of a Fig Tree on Rabies Virus.**] *C. R. Acad. Sci.* 1947, May 28, v. 224, No. 21, 1467–8.

The authors tested an emulsion of the filtered latex of the fig tree, *Ficus carica*, on the same experimental lines as in the case of agave juice [this *Bulletin*, 1947, v. 44, 950]. The titre of the latex was from 1/10 to 1/50, and at a *p*H of 7·2, under conditions and with controls comparable with those in the agave experiments, the fig juice destroyed a 1/50 rabbit-brain emulsion of the rabies virus *in vitro*. All the mice inoculated with the mixture of fig juice and virus survived, whilst those controlled with virus and water died of rabies. *H. J. O'D. Burke-Gaffney.*

BARNHART, J. M. **Review of the Literature and Report of a Case of Rabies.** *Texas Reports on Biol. & Med.* 1947, v. 5, No. 2, 209–20. [19 refs.]

MALARIA.

BASU, B. C. **The Frequency of Distribution of Gametocytes of the Indian Strains of Malaria Parasites.** *Indian J. Malariology.* 1947, Mar., v. 1, No. 1, 123–7.

"The frequency of distribution of gametocytes of Indian strains of three species of Plasmodium was studied in 318 gametocyte-carriers *P. vivax*, 84; *P. malariae*, 71; and *P. falciparum*, 163.

"The number of gametocytes of *P. vivax* varied between 'scanty' (less than 40) and 3,800 per c.mm., average 334; that of gametocytes of *P. malariae* varied between 'scanty' and 480 per c.mm., average 81, and that of gametocytes of *P. falciparum* varied between 'scanty' and 4,920 per c.mm., average 489."

JOSEPHSON, E. S., CULWELL, W. B. & TREMBLEY, Helen L. **Use of Aerosol OT in dissecting Salivary Glands of Mosquitoes infected with Malaria.** *Science.* 1947, June 20, 650–51.

Surface tension often interferes with easy manipulation of mosquitoes during dissection, and various methods have been employed to overcome this difficulty. Preliminary immersion in 70 per cent. alcohol is fairly effective, but a 1 : 6,000 dilution of Aerosol OT (dioctyl sodium sulphosuccinate) in physiological saline gives better results and dissection is carried out in this solution. The method, however, should not be employed if viable sporozoites are required, because Aerosol OT has a deleterious action upon them, though it does not interfere with their detection. *P. C. C. Garnham.*

JEPSON, W. F., MOUTIA, A. & COURTOIS, C. **The Malaria Problem in Mauritius; the Bionomics of Mauritian Anophelines.** *Bull. Entom. Res.* 1947, May, v. 38, Pt. 1, 177–208, 8 figs. [16 refs.]

Malaria first became evident in Mauritius in 1865, since when it has been widespread and at times epidemic. The anophelines responsible are *Anopheles funestus* and *A. gambiae*. The former is the most important vector and always associated with high splenic indices ranging up to 100 per cent., whilst *A. gambiae* alone is usually associated with splenic indices of the order of 40 per cent. This reversal of the common rôles is due to the lower temperature

tolerances of *A. funestus*, which can breed freely and transmit malaria for most of the year, whereas the free multiplication of *A. gambiae* is restricted by the temperature to four or five months of the hot wet season, with a consequent lessening of the severity of malaria. Both of these species become rare at altitudes of over 1,000 feet, at about which level malaria in consequence disappears.

Other factors affecting the incidence and seasonal distribution of malaria include rainfall, which operates both by extension of breeding places and by flushing of watercourses to give a variable effect; humidity, which is adequate for prolonged anopheline survival in the wetter lowlands, but rarely optimal in the drier north; and geological conditions which favour or impede the formation of breeding places. Throughout most of the island the soil is very porous and casual water persists for short periods only. In some areas of newer lava flow, however, seepages and springs are abundant, and there are a number of well-defined areas of impervious grey alluvial soil that call for special drainage measures.

A number of experiments on the ecology of *A. gambiae* and *A. funestus* are recorded; they concern chiefly the tolerance of larvae to different hydrogen ion concentrations, to salinity, to light, and to temperature. *A. gambiae* has a very marked tolerance to salinity. Extreme instances include development in waters with salt contents above that of sea water and breeding is common in coastal swamps with sodium chloride contents varying from 0·1 to 23 grammes per litre, a range covered by marshes supporting a growth of *Typha gravanica*, *Acrostichum aureum*, and *Paspalum vaginatum*, but usually exceeded by pure stands of mangrove except on their proximal edges where they are diluted by seepages or storm water channels. *A. funestus* has a much lower tolerance and is rare where the chloride content exceeds 2·0 grammes per litre.

The temperature of breeding places and the pace of larval development are related to each other and there is little doubt from the evidence given that rapid larval development of *A. gambiae* only occurs at quite high temperatures such as 30°C., the pace being greatly retarded by decreases of temperature to 25° and 20°C. From the data the authors deduce a mathematical formula showing the relationship and draw a curve, or two curves, described as symmetrical catenaries. [The non-mathematical reviewer had to look up the meaning of catenary, the form adopted by a chain suspended at both ends, but felt impelled to supply from his own knowledge the term extrapolation together with a warning against its dangers.] The apparent effect of shade in reducing breeding of *A. funestus* is almost certainly due to the lowered temperatures rather than to critical reductions in light intensity.

Melanism was found in larvae of *A. gambiae* in nearly all breeding places in the coastal belt, but in no case was 100 per cent. melanism observed, there was no relationship to degrees of salinity, and so far as is known, melanic larvae give typical *gambiae* adults, and there was no evidence of the presence of the variety *melas* in specimens sent elsewhere for identification. *G. Macdonald.*

RAO, V. V. & PHILIP, M. I. **The Relationship between the Breeding of *A. fluviatilis* and Distance from Human Dwellings.** *Indian J. Malariology.* 1947, Mar., v. 1, No. 1, 37–41.

" (1) *A. fluviatilis*, *A. minimus* and *A. varuna* breed in significant numbers up to at least half a mile from the place where they feed, and this distance is presumably their usual flight range in search of food.

" (2) Anti-larval operations against these species cannot safely be limited to less than half a mile in these areas."

RACHOU, R. G. & FERREIRA, M. O. Algumas observações sobre o indice larvário de anofelinos do sub-genero *Kerteszia* em bromeliáceas e sua densidade larvária no sul do Brasil. [**Observations on the Larval Index and Larval Density of Anophelines of the Subgenus *Kerteszia* in Bromeliads in the South of Brazil.**] *Folha Med.* 1946, Oct. 25, v. 27, No. 20, 9 pp. English summary.

These observations were made in 24 localities in the State of Santa Catarina and 6 localities in the State of Rio Grande do Sul in which anophelines of the subgenus *Kerteszia* breed in bromeliads. Altogether 12,538 bromeliads were examined; larvae were found in 1,724. In bromeliads growing on the ground, and epiphytic bromeliads less than two metres above ground, larvae were found in 16·4 per cent.—304 larvae per 100 positive bromeliads. In epiphytic bromeliads between 2 and 5 metres above ground, larvae were found in 13·4 per cent.—290 larvae per 100 positive bromeliads. In epiphytic bromeliads more than 5 metres above ground, larvae were found in 11·7 per cent.; 245 larvae per 100 positive bromeliads. *Norman White.*

RACHOU, R. G. Da infectibilidade dos anofelinos do sub-gênero *Kerteszia* pelos parasitas da malária humana. [**Infectibility of Anophelines of the Subgenus *Kerteszia* with Human Malaria Parasites.**] *Folha Med.* 1946, Dec. 5, v. 27, No. 23, 10 pp. [19 refs.] English summary (6 lines).

The author passes in review the considerable literature dealing with the capacity of anophelines of the subgenus *Kerteszia* to transmit malaria. In 1943, the National Malaria Service embarked on a programme of study of these species in southern Brazil. The author has found numerous naturally infested *A.* (*K.*) *cruzi*, *A.* (*K.*) *bellator* and (probably) *A.* (*K.*) *homunculus* in several localities in the State of Santa Catarina, where they are efficient vectors of malaria. *Norman White.*

SPITZ, Sophie. **The Pathology of Acute Falciparum Malaria.** *Milit. Surgeon.* 1946, Nov., v. 99, No. 5, 555–72, 32 figs. (6 coloured). [26 refs.]

This account is based on the " first fifty consecutive cases " of fatal *P. falciparum* malaria studied at the American Army Institute of Pathology during the recent war. The findings on the whole were similar to those of most other workers, but there were some points upon which new information was obtained. For instance, in the study of the brain lesions, Dr. Spitz found that " plugged " vessels were " not obstructed by parasitized erythrocytes but simply filled and distended by them, as if during life they were part of a moving stream." In cases showing haemorrhage and " granulomas ", however, thrombi were found in the " plugged " vessels, including the central arteriole of the " granuloma ", and thrombi were occasionally observed in the cortical capillaries. The thrombi were small and often did not completely occlude the lumens of the vessels concerned. The conception of vessels filled, but not obstructed, by parasitized cells is similar to that put forward by Rigdon (1942) in order to explain the apparently reversible nature of the cerebral changes in *falciparum* malaria. In the kidney, pigmented casts were observed in the distal convoluted lobules in 30 cases and " hemoglobinuric nephrosis ", similar to that seen in blackwater fever, was present in seven. In four of the latter cases and in five others, changes were observed in the glomeruli, including " generalized ischaemia, enlargement and increased cellularity." These cases had clinical evidence of azotaemia. The author considers that the incidence of haemoglobinuric nephrosis was unexpectedly high " since there were not other factors except the malaria itself by which to explain this phenomenon." She holds that glomerular

dysfunction is an important factor in the pathogenesis of the renal insufficiency observed, and that the nephrosis, together with the myocardial cellular infiltration and adrenal atrophy also observed, provide strong evidence in favour of "circulating toxins or allergens" liberated by *P. falciparum.* Selective localization of parasites in the various organs could not explain the pathogenesis of the lesions. Thrombosis could be demonstrated only in some of the small cerebral vessels; it was not found in other organs even where, as in the heart, "plugging" with parasitized cells was prominent.

The illustrations in the article are excellent and the descriptions remarkably clear. *B. G. Maegraith.*

SCHNEIDER, L. A. & SHALLENBERGER, P. L. **A Study of Hepatic Function in Acquired Malaria.** *Amer. J. Med. Sci.* 1947, July, v. 214, No. 1, 80–83, 2 figs.

In an attempt to detect liver damage due to malaria, 250 patients suffering from pure *P. vivax* infections were subjected to various tests. These tests included estimations of bromsulphthalein retention, icteric index, hippuric acid synthesis, prothrombin time, serum total protein and albumin/globulin ratio, and the cephalin-cholesterol flocculation test. The tests were applied as soon as the patients had become afebrile under treatment with a standard course of mepacrine, which was usually within two to three days of the commencement of therapy. According to the more specific tests of liver function approximately one-third of the patients showed evidence of liver damage, while the less specific tests were positive in about two-thirds of the patients. Repetition of the tests suggested that hepatic dysfunction associated with malaria is transitory and that permanent liver damage is rare, except possibly where the liver is already diseased. *F. Murgatroyd.*

HENRY, A. F. X. La recherche de l'activité plasmodiale au cours de la prémunition et du paludisme chronique. [**Signs of Activity of the Malaria Parasite in Patients in a state of Premunition and in Chronic Malaria.**] *Bull. Acad. Nat. Méd.* 1947, v. 131, Nos. 11/12, 216–19.

In countries in which malaria is endemic, the state of premunition which many of their inhabitants enjoy may weaken in certain conditions. A superadded morbid condition may then reawaken malaria activity, though the parasites may not be discoverable in blood smears. The author details the clinical signs and laboratory tests on which he relies to evaluate the rôle of malaria in the causation of the patient's condition.

Melano-flocculation may lead one to suspect the persistence of a malaria infection that was believed to have disappeared. Quantitative and qualitative changes in blood albumin are significant. The Takata Ara reaction is of great value; the author considers as positive flocculation in at least three tubes including the fifth tube, 1/32 dilution. Flocculation in only two tubes is an indication for a repetition of the test. Determination of bilirubin in the blood should not be forgotten. Blood urea may be increased, but it is often below normal. Cholesterin is sometimes increased but often notably diminished. A notable increase in the red cell sedimentation rate is an important sign. Differential blood cell counts give important indications.

Pathological conditions are sometimes attributed to malaria without sufficient proof: it is none the less true that an unsuspected malaria infection is responsible for many failures of treatment. An interesting case of cirrhosis of the liver in an alcoholic is described. The prognosis was most unfavourable. A positive melano-flocculation test was obtained. Antimalarial treatment contributed to an unexpected recovery. *Norman White.*

STUDDERT, T. C. **Subtertian Malaria in White Troops in West Africa. A Review of 300 Cases.** *J. Trop. Med. & Hyg.* 1947, Mar., v. 50, No. 3, 44–52, 2 charts. [15 refs.]

LAMBILLON, J. Lésions érésipéloïdes d'origine malarienne. [**Erysipeloid Lesions of Malarial Origin.**] *Ann. Soc. Belge de Méd. Trop.* 1947, Mar. 31, v. 27, No. 1, 91–3.

The author records 7 cases, four of which are described in detail, in which lesions resembling erysipelas were associated with malaria. In most of the cases the lesion was a red patch showing characters apparently typical of erysipelas: the temperature was high, but the absence of a significant leucocytosis was perplexing. It was not, indeed, until the first two cases had been studied that the author was able to identify the condition at an early stage in the other five. All the lesions occurred on the lower limbs. In the first two cases, the diagnosis of erysipelas was considered, but malaria parasites were found in the blood. The patients were treated with mepacrine and "Prontosil" and the acute condition cleared in a few days, leaving a slowly regressing ecchymosis.

In the third case, the patient had been treated for erysipelas, apparently with success, by sulphonamides before he was seen by the author, by which time he had showed a similar lesion. His blood showed only 9,000 leucocytes per cmm. and malaria parasites were present. The condition resolved as in the previous cases, but later the temperature rose and a similar lesion occurred on the other limb. On this occasion, the leucocytosis varied from 23,000 to 27,000 cells per cmm., no parasites were present in the blood and the case was evidently one of genuine recurring erysipelas, which on one occasion had shown an identical lesion during an attack of malaria.

The last case occurred in a patient who had an attack of malaria, with slightly raised temperature (he had been taking quinine regularly), and the cutaneous lesion described seemed to be intermediate between a purpuric one and the erysipeloid lesion seen in the other cases.

The author suggests that these cutaneous lesions are provoked partly by emboli of infected red cells or parasites in the capillaries and partly by lowered local capillary resistance in those areas where the circulation is relatively limited. *H. J. O'D. Burke-Gaffney.*

BUTLER, F. A. & SAPERO, J. J. **Pacific Vivax Malaria in the American Negro.** *Amer. J. Trop. Med.* 1947, Mar., v. 27, No. 2, 111–15, 1 fig.

In 1942–43 a United States force composed of 72 per cent. white and 28 per cent. Negro troops were on a malarious island in the South Pacific. The Negroes were mostly from the Carolinas and Georgia and were aged from 20 to 35 years. Malaria incidence statistics collected on an advanced base were inevitably incomplete, but they are sufficient to justify the conclusion that the Negroes had no greater tolerance to the strain of *P. vivax* malaria encountered on that South Pacific island than had the white troops.

[This experience is in marked contrast to the numerous unsuccessful attempts to infect Negroes with Pacific strains of *P. vivax* that have been reported from the United States.] *Norman White.*

BIANCO, A. A., SAUNDERS, G. M., LEVINE, A. S. & COHN, R. **Long-Term Observation of *Plasmodium vivax* Malaria in the Returned Serviceman. Part I.** *U.S. Nav. Med. Bull.* 1947, Mar.–Apr., v. 47, No. 2, 352–67. [19 refs.]

A clinical test of new antimalarial drugs necessitated the observation of a large number of patients for periods of from one to nine months. Advantage

was taken of the opportunity thus afforded to study the clinical features and any unusual sequelae of malaria. The results of this study are recorded. About 1,500 attacks of malaria were observed in marines and naval men. All were due to *P. vivax*; the patients had previously been on more or less regular atebrin suppression.

In addition to the clinical picture and positive smear certain laboratory findings are consistent with a diagnosis of malaria, namely, a leucopenia with increase of mononuclear cells, increased erythrocyte sedimentation rate, and an otherwise unexplained positive Kahn reaction.

A few true delayed primary attacks were seen. Such attacks were insidious in onset. They may be treated as pneumonia or some catarrhal fever before the true nature of the disease is determined. The diagnosis is sometimes made more difficult because of the empirical use of the sulphonamides in such conditions. Malaria cachexia and the chronically enlarged spleen were not observed in this series of cases.

The complement fixation test, with *P. gallinaceum* as antigen, was of no aid in the diagnosis of the acute attack.

A large proportion of cases of malaria gave positive serological reactions with most tests in use for the diagnosis of syphilis. The percentage of positive reactors increases during the second and third weeks after the onset of the clinical attack, but tests are generally negative within four weeks, and always within ten weeks, after onset. The duration of clinical activity appears to have little influence upon the persistence of positive reactions.

Norman White.

MAROUN, T. L'action du traumatisme sur les rechutes paludéennes. Considérations médico-légales et médico-sociales. (Déductions thérapeutiques.) [**Trauma as a Cause of Malaria Relapse : Medico-legal and Medico-social Considerations.**] *J. Roy. Egyptian Med. Ass.* 1947, Feb., v. 30, No. 2, 86–98.

The author discusses the rôle of trauma in reactivating latent malaria, from the point of view of forensic medicine. He describes three cases from his experience in which criminal assault precipitated attacks of malaria. One patient died of malaria. Another committed suicide while suffering from delirium attributed to malaria. The third recovered. The question is raised as to whether the assailant should be held guilty of having caused the death of his victim in the two fatal cases ; in neither case were the wounds of sufficient gravity to have caused death, had it not been for the malaria infection. The penal codes of different countries differ in this matter. *Norman White.*

MAKOWER, H. **Observations on the Beneficial Effect of Blood Transfusions on Relapses in Benign Tertian Malaria.** *Texas Reports on Biol. & Med.* 1947, v. 5, No. 2, 185–7.

Malaria became frequent in Poland during the late war, because of movements of troops and civil populations. Antimalarial drugs were difficult to obtain ; only quinine was available and some patients responded poorly to it. Blood transfusions were therefore given in some cases with the aim of influencing the course of the disease.

Two such cases in soldiers are described in full. Both patients had chronic malaria, with bloody diarrhoea. Their attacks of malaria increased in severity and frequency and the patients became resistant to quinine. Anaemia of the pernicious type developed. Blood transfusions of 250 cc. of stored blood were given and repeated a few days later (in one case increased to 500 cc. : in the

other, repeated and then increased to 500 cc. in a third transfusion two weeks later). This resulted in rapid clinical and haematological improvement, but parasites persisted in the blood. In one case, atebrin (mepacrine) and plasmoquine and, in the other, mepacrine only, rendered the blood free of parasites. It was evident that the beneficial effect of the blood transfusions brought about a " stimulation of the defence mechanisms ", but the disease was transformed from a manifest to a latent one, requiring specific drug therapy.

The author was unable to determine whether the cases were really quinine-resistant or whether the quinine was not absorbed properly by an intestine damaged by a prolonged diarrhoea. *H. J. O'D. Burke-Gaffney.*

WISELOGLE, Frederick Y. **A Survey of Antimalarial Drugs 1941–1945.**

This book is reviewed on p. 1106.

HAGGIS. **Fundamental Errors in the Early History of Cinchona.** *Bull. History of Med.* 1941, Oct. & Nov., v. 10, Nos. 3 & 4, 417–59 ; 568–92, 11 figs. & 1 map. [Refs. in footnotes.]

BARANGER, P. Revue générale de la question des totaquinas. [**A Review of Totaquina.**] *Rev. Paludisme et Méd. Trop.* 1947, July–Aug.–Sept., v. 5, Nos. 38–39–40, 201–12.

TRAGER, W., BANG, F. B. & HAIRSTON, N. G. **The Effect of Four Different Therapies on the Relapse Rate of Vivax Malaria.** *Amer. J. Hyg.* 1947, Jan., v. 45, No. 1, 43–57, 4 figs.

These observations were made in a reconditioning camp established by the U.S. Army in a non-malarious part of Australia to which men who had acquired malaria in New Guinea were sent. A comparison was made of the relapse rates after four different methods of treating the clinical attack of *P. vivax* malaria. The treatments were : (1) Atebrin 1·2 gm. during the first 24 hours ; 0·8 gm. during the second 24 hours, and 0·4 gm. daily for the next four days : total 3·2 gm. in 6 days. (2) Treatment as (1) followed by plasmoquine hydrochloride 0·02 or 0·03 gm. a day for five days. A suppressive dose of atebrin 0·1 gm. was also given during these five days, atebrin after one meal, plasmoquine after the other two meals. (3) Treatment as (1) followed by sulphamerazine 3 or 4 gm. a day for five days. Here also suppressive atebrin was given, 0·1 gm. a day, during the five days. (4) Quinine 3·0 gm. a day for the first three days, then 2 gm. a day for three days. Following treatments (1), (2) and (3) suppressive atebrin 0·1 gm. each day except Sundays was continued for at least three, usually four to six, weeks. After treatment (4) quinine 0·6 gm. a day was given for at least three weeks.

With treatments (1), (2) and (3) no relapse occurred during suppressive treatment ; the peak incidence of relapses was 4 to 5 weeks after discontinuance of suppression. This time interval was not influenced either by the maximum atebrin plasma level observed during treatment, or the length of the period of atebrin suppression. In the quinine group, some relapses occurred while the men were taking suppressive quinine.

The overall relapse rates during a period of 4 to 7 weeks or longer after suppression were : atebrin 76 per cent. ; atebrin-plasmoquine 61 per cent. ; atebrin-sulphamerazine 69 per cent. ; quinine 82 per cent. The differences in the relapse rates following the three atebrin treatments were not statistically significant. *Norman White.*

BUTLER, Ruth, DAVEY, D. G. & SPINKS, A. **A Preliminary Report of the Toxicity and the Associated Blood Concentrations of Paludrine in Laboratory Animals.** *Brit. J. Pharmacol.* 1947, Sept., v. 2, No. 3, 181–8, 3 figs.

In these experiments, paludrine was used as the monoacetate and monohydrochloride, the former for intraperitoneal and intravenous and the latter, which is less soluble, for oral tests. Both salts are stable in boiling aqueous solution. The largest dose which allowed all animals to survive (LD0) was determined as well as LD 50 and LD 100. In rats and mice, but not in chicks, death was delayed after administration by the first two routes. A second intravenous dose some hours after the first produced an additive toxic effect in the mammals. Estimation of the concentration of drug present showed that the delayed deaths in rats and mice were not due to its retention in the blood, and it is possible that in their case a more toxic substance is formed from paludrine as the result of metabolic processes. In chronic toxicity tests, blood concentrations were estimated and the effect of different dosages on the growth curves determined. No significant pathological lesions were found and the central nervous system appeared to be unaffected. The blood reached the brain most readily in the case of chicks. Comparison of blood concentrations in man and rats showed that a particular level in the latter was associated with greater toxicity than in the former, in whom the drug level was better maintained. In chicks, which tolerate a great concentration of drug in the body, the blood level of paludrine associated with lethal effects was about 10 times that in rats and mice. The distribution of the drug did not account for differences in toxicity. The behaviour of paludrine in man, to whom it is but little toxic, resembles more closely that in the chick than it does that in the rat or mouse. *J. D. Fulton.*

PATIÑO CAMARGO, L., CORTÉS, E., GARCÍA, V., SANTOS, J., ZOZAYA, C., RUEDA V. & ROSA DEL VALLE, Julia. Información sobre la droga W7618, Difosfato 7-Cloro-4 (4-dietilamino-1-metilbutilamino) quinolina, en el tratamiento del paludismo. [**Information concerning the Drug W7618, Diphosphate 7-Chloro-4 (4-diethylamino-1-methylbutylamino) Quinoline, in the Treatment of Malaria.**] *Rev. Facul. de Med.* Bogota. 1946, Oct., v. 15, No. 4, 261–3.

The authors treated sixteen patients, suffering from malaria, with compound W7618 (Winthrop, New York). Eleven were chronic relapsing cases and five had primary acute attacks. Five had *P. vivax*, six had *P. falciparum* and five had mixed infections. The results were satisfactory. The drug is effective and easy to handle; the treatment is short, 3 days; no toxic phenomena of any consequence were noted; there was no discolouration of the skin.

Norman White.

CHAPMAN, N. B., GIBSON, G. M. & MANN, F. G. **Synthetic Antimalarials. Part XVI. 4-Dialkylaminoalkylaminoquinazolines. Variation of Substituents in the 6- and 7-Positions.** *J. Chem. Soc.* 1947, July, 890–99.

CURD, F. H. S., RAISON, C. G. & ROSE, F. L. **Part XVII. Some Arylaminoaminoalkylaminoquinoline Derivatives.** *Ibid.* 899–909.

MANN, F. G. & Porter, J. W. G. **Part XVIII. 3-Dialkylaminoalkylaminodiphenylamines.** *Ibid.* 910–14.

MANN, F. G., NAYLOR, F. T. & PORTER, J. W. G. **Part XIX. Dialkylaminoalkylaminodiphenylguanidines.** *Ibid.* 914–17.

KENEFORD, J. R. & SIMPSON, J. C. E. **Part XX. Cinnolines. Part XIII. Synthesis and Antimalarial Action of 4-Aminoalkylaminocinnolines.** *Ibid.* 917–20.

BANG, F. B. & HAIRSTON, N. G. **Studies on Atabrine (Quinacrine) Suppression of Malaria. III. The Epidemiological Significance of Atabrine Suppression.** *Amer. J. Trop. Med.* 1947, Jan., v. 27, No. 1, 31–8, 2 figs. [12 refs.]

The authors relate the malaria experiences of two divisions in the South Pacific, during and after combat. Determination of atebrin blood levels, thick smear surveys, mosquito dissections and malaria surveys were carried out. They conclude that atebrin suppression not only suppresses individual attacks of malaria, but also has a damping effect on the development of epidemic malaria in troops. The malaria rate in troops, the gametocyte rate in troops and the infection rate in mosquitoes vary in direct relation. Adequate atebrin suppression prevents many infections that would otherwise take place. The routine administration of plasmoquine in the treatment of the acute attack is of little practical value in preventing transmission among troops. None of four *P. falciparum* carriers discovered in routine surveys had ever been treated for malaria. It is possible during imperfect suppression to follow malaria transmission by observing the ratio of *falciparum* to *vivax* malaria.

Norman White.

HOCKING, K. S. **Assessment of Malaria Control by Mosquito Prevalence.** *Bull. Entom. Res.* 1947, May, v. 38, Pt. 1, 131–6, 3 figs.

Assessment of the number of cases of malaria, though desirable, gives too delayed and too unreliable a picture of the efficiency of control, and hand catching of mosquitoes in "catching stations" is preferable. In East Africa, systematic catching in and around controlled areas gives a very reliable picture of the efficacy of anti-mosquito measures. The technique is not normally standardized and is open to some objections, to meet which the author has devised a portable standard trap hut, from catches in which uniform results may be expected.

G. Macdonald.

JOHNSON, H. A. & YATES, M. W. **DDT in Oil as a Larvicide in an Area ordinarily considered difficult to treat.** *Pub. Health Rep.* Wash. 1947, July 25, v. 62, No. 30, 1085–95, 2 figs.

The sites in question were difficult to treat because of the large areas of water, difficult to traverse and heavily overgrown. Instead of relying on plain oiling at the rate of 23 (U.S.) gallons per acre, the authors found it satisfactory to apply 9 to 13 gallons of 1 per cent. DDT, or 6 to 7 gallons of 2 per cent. DDT, in oil, for the control of *Anopheles* breeding [species not stated]. Spraying was done at the same intervals and with the same apparatus, but resulted in considerable saving of man-hours. The rates of DDT applied, however, were of the order of 1 lb. per acre. This was dangerous to fish and other wild life and such applications can only be recommended in areas where this risk is unimportant. [1 U.S. gallon equals 4/5 U.K. gallon.]

J. R. Busvine.

HADAWAY, A. B. & BARLOW, F. **Insecticidal Effect of Surface Deposits of D.D.T. on Mud.** [Correspondence.] *Nature.* 1947, Sept. 13, 363.

Field experiments in malaria controls have demonstrated that, in residual spraying of houses with DDT, the surface deposit applied must be such as to be lethal to mosquitoes after only a brief contact. Since the common types of surface in African houses are the mud wall and the thatch roof, the authors, working in the Colonial Insecticide Research, Uganda, have conducted laboratory tests to determine the efficacy of different formulations of DDT on mud.

Standard mud blocks, 2 in. square by 1 in. deep were made from sun-dried soil passed through a 40-mesh sieve. The blocks were dried slowly in wooden frames at room temperature.

Five per cent. DDT (*pp'*) was applied to 5/4 blocks with a Four Oaks "Kent" sprayer in dosages from 100 to 400 mgm. per square foot; toxicity tests were carried out 24 hours later on *Glossina palpalis* and *Aëdes aegypti*.

The outer layer of each set of four blocks was then scraped off and made up to 1 gm. in weight (representing a layer of about 0·1 mm. thick). The deposit of insecticide in this layer and in the whole blocks was determined chemically. This outer layer contains more than all the contact available insecticide. There was considerable absorption of oil solutions by the mud and only 6 to 15 per cent. of the insecticide appeared in the outer layer: mortality of the test insects was low, even with dosages of 1,000 mgm. per square foot.

The highest proportion of insecticide recovered from the outer layer (62 per cent.) was from blocks treated with a water suspension of dispersible powder, the suspended particles being filtered off at the surface. There was 100 per cent. mortality of *G. palpalis* after 15 sec. contact and up to 90 per cent. mortality of *A. aegypti* after half an hour.

Comparative results of different formulations are given in a table, which indicates the superiority of the dispersible powder. Initially promising results with a "Gammexane" dispersible powder 530 were not maintained when the percentage of insecticide was determined 10 days later. Detailed results of these tests and of field trials now in operation will be published.

H. J. O'D. Burke-Gaffney.

MORGAN, R. E., SHEPPARD, R. R. & BARNETT, G. S. **Malaria Control. Description of a Compressed Air Sprayer.** *U.S. Nav. Med. Bull.* 1947, May–June, v. 47, No. 3, 529–35, 4 figs.

In order to treat vegetation (for adult anophelines) and pools (for larvae) in jungle areas, a large insecticide sprayer mounted on a lorry is used. A 1½ ton truck carries an air compressor producing 210 cu. ft. of air per minute. This is used to atomize a 5 per cent. solution of DDT in Diesel oil from ten horizontal nozzles mounted behind the lorry. The output moving at 4 m.p.h. gives 0·1 lb. of DDT per acre. The mist produced consists of rather large droplets of various sizes (7 to 1,300 microns diameter, averaging about 100–500). Larvae in breeding sites up to 500 ft. from the point of emission were killed in 2 hours and at ½ mile they were killed in 48 hours.

J. R. Busvine.

HEATH, R. F. **Sanitation. An Experiment in Mosquito Control by Air-borne Sprays.** *Canadian J. Pub. Health.* 1947, Aug., v. 38, No. 8, 405–7.

WILSON, D. Bagster & ROBERTSON, A. G. **DDT Airspray in Malaria Control in East Africa.** *Trans. Roy. Soc. Trop. Med. & Hyg.* 1947, July, v. 40, No. 6, 823–50, 10 figs.

Airspraying makes the control of otherwise inaccessible mosquito breeding places possible, although, at the present price of DDT, it is not an economic alternative to larvicidal ground control when that is possible and equally effective. Ground control may be impossible either because there is some impassable barrier, usually water, or because of the great extent of the breeding area to be covered. Of the two causes, the extent of breeding areas is more

likely to lead to economic applications of airsprays, for under most circumstances it should prove cheaper and more efficient than the institution of large capital works. The value of airspray in wartime has been proved, and it should prove of similar value in controlling an epidemic of malaria. The possible economic disadvantages of the widespread distribution of DDT remain to be explored, but the authors show that a single spraying at a dosage of 32 mgm. per square metre had no effect on fish and only a slight effect on plankton, but that seven sprayings at a similar concentration caused an undoubted mortality amongst fish and aquatic insects over a limited area. They consider, however, that spraying over a limited area of open lake would be unlikely to have any appreciable effect on fisheries as a whole.

The sprayings began some two weeks ahead of the normal period of maximal rainfall and the DDT (4 or 5 per cent. in oil) was delivered from a 300-gallon bomb-bay tank through a single 2·5-inch emission pipe ; the rate could not be varied and was about 2 gallons per second.

Owing to the fact that the Service populations were being given suppressive mepacrine, the means of assessment of the effectiveness of the sprayings was entirely limited to entomological observations. These observations were made at a series of stations which were outside, or within, the sprayed area. At these stations, all available adult mosquitoes were caught and identified at regular intervals.

Larval sampling consisted in making a set number of dips per station, with ladles, the larvae subsequently being counted and classified by stages and genera.

The authors' summary is as follows :—

" 1. An account is given of the use of DDT airspray against *Anopheles gambiae* and *funestus* in East Africa.

" 2. The entomological assessment of the series of sprayings carried out shows that spraying at a dosage of 32 mg. per sq. metre of pure DDT gave an immediate reduction of *Anopheles* of 98 per cent. ; but that a dosage of 17 mg. per square metre gave results that were little better than standard ground control methods.

" 3. The effectiveness of these two rates of application followed closely the observed larvicidal action, but there was an unexplained immediate effect on adult anopheline population at the higher dosage.

" 4. Although this was not conclusively demonstrated, it is concluded that the seasonal peak of *gambiae* breeding was effectively, if not completely, checked, this conclusion being based on the slow rate of recovery of vector house catches, and on comparison with previous years.

" 5. Some substantial evidence is provided that the radius of control against these two species need not in practice be extended, in the fairly typical localities under discussion, beyond 1½ to 2 miles.

" 6. The main applications of airspray arise out of the speed and simplicity of its initiation and practice (granted the necessary familiarity with the method), and the capacity of aircraft to overcome ground inaccessibility.

" 7. Reasons for the comparatively high dosage shown to be necessary were found in the interference to the penetration of the spray to the water surface by vegetational cover, and in the variability of spray distribution attributable to variations in windspeed and direction and to wind turbulence.

" 8. A further cause for uncertainty in distribution, by the method used, lies in the nature of the drop spectrum of the spray, and this can probably be overcome by wide overlapping of swathes at a reduced rate of spray emission.

" 9. It is considered that airspraying over the limited areas to be covered in practical malaria control is unlikely to cause any serious upset of the biological balance of other fauna."

R. M. Gordon.

RIBBANDS, C. R. **The Use of Residual Films of DDT and Gammexane in Malaria Control.** *Bull. Entom. Res.* 1947, Mar., v. 37, Pt. 4, 567–92, 4 figs. [13 refs.]

This work was carried out in Assam, where malaria is carried by *Anopheles minimus*, with the object of developing an efficient and economical residual spray technique, of comparing the efficiency of different sprayers, of the two insecticides available (DDT and benzene hexachloride) and of different doses and types of application.

The sprayers tested were :—(*a*) motor-driven air pressure sprayers of the paint gun type ; (*b*) " MISH " (Malaria Institute of India) flit guns ; (*c*) Sheltox flit guns ; (*d*) Four Oaks Knapsack oil sprayers ; (*e*) Solo sprayers ; (*f*) Stirrup pumps ; and (*g*) hand-operated knapsack pressure sprayers of the type pumped up before use. The first three, in which the spray is atomized by air, were all considered too wasteful for normal use. All the others could be used satisfactorily the last having fewest disadvantages. Greatest efficiency was secured when the nozzle was held at about one foot from the wall to be treated.

On theoretical grounds, the author believes that an observed 90 per cent. reduction in vector anophelines almost certainly indicates complete control of malaria transmission, and a 70 per cent. reduction a very probable elimination of the disease, and his results, tabulated in detail, are illustrated on this assumption. The standard dosage employed was one quart of solution to 200 sq. ft., the quantity of insecticide applied being varied by modification of the strength of solution [one quart of 5 per cent. DDT to 200 sq. ft. is equivalent to 10 oz. DDT per 1,000 sq. ft.]

The periods for which DDT solution in kerosene gave a continuous 90 per cent. reduction in *A. minimus* were :—10 per cent. solution over 20 weeks ; 5 per cent. over 20 weeks ; $2\frac{1}{2}$ per cent., 8–12 weeks ; $1\frac{1}{4}$ per cent., 12 weeks ; $\frac{5}{8}$ per cent., 5 weeks ; $\frac{5}{16}$ per cent., 1 week. DDT/oil/water emulsions gave similar control for the following periods :—10 per cent. over 20 weeks ; 5 per cent., 12–15 weeks ; $2\frac{1}{2}$ per cent., over 20 weeks ; $1\frac{1}{4}$ per cent., over 20 weeks ; $\frac{5}{8}$ per cent., 10 weeks ; $\frac{5}{16}$ per cent., 3 weeks. The emulsion was therefore considered superior to the solution, and heavy doses were thought preferable, on account of their long duration of effect, to smaller ones.

Gammexane was used in the form of crude benzene hexachloride containing 12 per cent. of the gamma isomer. The duration of effect of gammexane solutions in kerosene against *A. minimus* was :—5 per cent. (0·6 per cent. gammexane) 7 weeks ; $\frac{5}{8}$ per cent. (0·075 per cent. gammexane) 4 weeks ; $\frac{5}{16}$ per cent. (0·0375 per cent. gammexane) 2 weeks. A 5 per cent. (0·6 per cent. gammexane) oil/water emulsion was effective for 10 weeks. All these trials were on a smaller scale than those with DDT.

In these trials, the author treated the complete inner surface of the houses. He also tried fractional treatment of the walls, applying 5 per cent. DDT solutions to the outside walls, the ceilings, inner walls, the lower half and the lower quarter of internal walls. Treatment of the inner walls only gave over 20 weeks complete control of *A. minimus*, of the ceilings 18 weeks, the others being relatively ineffective. A small series of trials of gammexane solutions on the eaves only, with a 5 per cent. solution (0·6 per cent. gammexane) gave 4 weeks' complete control, and $1\frac{1}{4}$ per cent. (0·15 per cent. gammexane) 3 weeks' control. Equivalent figures for DDT solutions were :—5 per cent., 8 weeks ; $1\frac{1}{4}$ per cent., 1–6 weeks.

The general conclusion drawn is that emulsions of DDT are superior to solutions, though there is no such disparity in the case of gammexane ; treatment of walls or ceilings only may be adequate, ceilings perhaps being easier and less subject to disturbance or re-surfacing. The author does not exclude the possi-

bility of repellent effects lessening the actual degree of malaria control achieved, but regards his figures as suitable guides to the type of treatment most likely to be successful. The rôle of gammexane remains unestablished; though the slight differences in its properties may give it some advantages when compared with DDT. *G. Macdonald.*

MADDEN, A. H., SCHROEDER, H. Ö. & LINDQUIST, A. W. **Residual Spray Applications to Salt-Marsh and Jungle Vegetation for Control of Mosquitoes.** *J. Econom. Entom.* 1947, Feb., v. 40, No. 1, 119–23.

Rather heavy doses (15 to 30 gallons per acre) of 5 per cent. DDT either in fuel oil, aqueous emulsion or suspension, were applied to marsh or jungle vegetation by the use of knapsack sprayers working on 6-foot swaths. The effects were estimated by the toxicity to flies and mosquitoes, of vegetation cut from the treated plots at various periods up to 60 days; and also by observations of mosquitoes caught in the treated and untreated areas. Additional tests were made with pyrethrum solution (1 per cent. pyrethrins) and benzene hexachloride emulsion (5 per cent. of BHC containing 12 per cent. *gamma* isomer). The DDT suspensions gave the best results, which ensured about 75 per cent. reduction for forty-six days. The BHC gave promising results, especially in the first three weeks, and might have been as good as the DDT at equal doses of active principle. *J. R. Busvine.*

HALL, T. F. & HESS, A. D. **Studies on the Use of 2,4-D for the Control of Plants in a Malaria Control Program.** *J. National Malaria Soc.* 1947, June, v. 6, No. 2, 99–116, 5 figs.

Investigations were made on the use of 2,4-D (2,4-dichlorophenoxyacetic acid; a growth-regulating substance) for the control of plants which create malaria control problems associated with breeding of *Anopheles quadrimaculatus* in Tennessee Valley Authority reservoirs. Detailed studies on 16 plant species and general observations on an additional 58 herbaceous and 24 woody species were made. Materials tested included 2,4-D dusts, triethanolamine salt and the sodium salt, methyl ester and butyl ester of 2,4-D; effectiveness was estimated by percentage changes in cover and density in treated as compared with untreated quadrats. In general, there were no great differences in control with the different formulations, although there was some indication that salts were more effective against cocklebur (*Xanthium americanum* Walt.) while the esters were more effective against lotus (*Nelumbo pentapetula* Walt.).

Dusts were applied in 10 per cent. 2,4-D concentrations in pyrax, at rates of 2·5 to 10 lb. 2,4-D per acre in hand applications; and 2·5 to 5 lb. per acre from airplanes. Sprays (aqueous solutions of the salts, kerosene solution of butyl ester, aqueous emulsion of methyl ester) were applied at dosages of 1 to 3 lb. per acre in concentrations of 0·5 to 5 per cent. 2,4-D in hand applications, 0·1 to 1·5 per cent. in ground and boat power equipment and 20 to 72 per cent. in airplane treatment. Details of spray equipment are given. It was found that 0·5 per cent. solutions gave more even cover and therefore better results than 5 per cent. solutions when applied by hand.

Certain species such as ragweeds (*Ambrosia* spp.), cocklebur, lotus and black willow (*Salix nigra* Marsh) were highly susceptible to 2,4-D while Burhead (*Echinodorus radicans*), giant ragweed (*A. trifida* L.), trumpet vine (*Campsis radicans* Seerman), water primrose (*Jussiaea diffusa* Forsk.) and wild cotton (*Hibiscus* spp.) were highly susceptible to the sodium salt, at least when "dewatered" and all were effectively controlled at dosages at 1 lb. 2,4-D per acre. Other species were highly resistant and difficult or impossible to control

with a single application (*Typha latifolia* L., *Zizaniopsis miliacea*, *Scirpus cyperinus* L. and button ball, *Cephelantus occidentalis* L.) and gave no reduction at 3 lb. per acre. Other species were intermediate in susceptibility; a considerable list of susceptible, less susceptible and resistant plants is given.

Airplane application appears to be the most satisfactory and economical method for area treatment against such species as lotus.

A higher recovery was observed with sprays of about 66 per cent. of material as compared with dusts (30 per cent.) in the centre 50 feet of the swath.

Such highly concentrated materials as 72 per cent. butyl ester, gave 100 per cent. kill over 112 feet swath, at an average dose of less than 1 lb. of 2,4-D and about 1 pint of liquid per acre. 10 per cent. dust at 2·5 lbs. 2,4-D per acre gave 60 per cent. reduction of cover after one application and a 90 per cent. reduction after an additional application was made 2 days later.

The results indicated that dusts were not so effective as sprays in equivalent dosages for lotus control.

On the basis of results obtained, the authors believe that the use of 2,4-D will provide a more efficient and economical means of control of terrestrial plants and of most stiff-stemmed annuals, when these create major malaria control problems, than control by mechanical means. It is pointed out, however, that some crop plants (*e.g.* cotton) may be highly susceptible to 2,4-D and that drift of small quantities a few hundred feet away from the site of application may have adverse effects on crops. *C. G. Johnson.*

i. McKee, R. W., Ormsbee, R. A., Anfinsen, C. B., Geiman, Q. M. & Ball, E. G. **Studies on Malarial Parasites. VI. The Chemistry and Metabolism of Normal and Parasitized** (*P. knowlesi*) **Monkey Blood.** *J. Exper. Med.* 1946, Dec. 1, v. 84, No. 6, 569–82, 2 figs. [36 refs.]

ii. Geiman, Q. M., Anfinsen, C. B., McKee, R. W., Ormsbee, R. A. & Ball, E. G. **VII. Methods and Techniques for Cultivation.** *Ibid.* 583–606, 5 text figs. & 4 figs. on 2 pls. [49 refs.]

iii. Anfinsen, C. B., Geiman, Q. M., McKee, R. W., Ormsbee, R. A. & Ball, E. G. **VIII. Factors affecting the Growth of** *Plasmodium knowlesi* **in Vitro.** *Ibid.* 607–21, 1 fig.

i. In the first paper are described the results of a biochemical investigation on the blood of normal rhesus monkeys as compared with that of monkeys having a *Plasmodium knowlesi* infection. The object of the study was to obtain information regarding the necessary constituents of a medium suitable for the *in vitro* cultivation of the malarial parasites. In the first place it became clear that an adequate supply of glucose would be essential. The normal erythrocyte of the monkey, like that of the human being, depends on glucose, which it converts into lactate. Therefore if normal erythrocytes are to be present in a culture medium they will require a supply of glucose to keep them in good condition. It is found, however, that the parasitized cell consumes glucose at a rate 68 times that of the normal cell, the actual rate being at any time roughly proportional to the parasite mass. Unlike the normal cell, the parasitized cell can make use of lactate at a rate which is one-sixth of that at which it is produced from glucose. It can do this only in the presence of oxygen. It was found that the pyruvate concentration tended to parallel that of the lactate, so that it seems justifiable to conclude that pyruvate is a temporary intermediate oxidation product. The ability of the parasitized erythrocyte to oxidize lactate indicates that the parasite synthesizes oxidative enzymes, which do not occur in normal cells. Also the fact that the parasitized cell converts glucose to lactate suggests that the parasite synthesizes enzymes

necessary for glycolysis. Direct measurements of dehydrogenase activity indicated that the cells containing half-grown parasites exhibit a dehydrogenase activity two to two and a half times that of normal cells. As the rate of conversion of glucose into lactate is far in excess of the rate of consumption of lactate by the parasitized cell, adequate *p*H control could only be accomplished by dilution of the parasitized blood with a buffered isotonic nutrient medium, or provision for the continual addition of glucose and removal of lactic acid from the whole blood by dialysis against a suitable nutrient medium. As regards the inorganic constituents of normal monkey plasma and erythrocytes, these are similar to those of human beings. In the case of parasitized blood the inorganic phosphate values for plasma are higher during segmentation of the parasites. Other inorganic components of parasitized blood are near the normal level. The parasitized cells consume oxygen in the presence of glucose, lactate, glycerol and amino acids. This consumption is inhibited by cyanide, carbon monoxide and high oxygen tensions. Methylene blue alone enables the normal red blood cells to consume oxygen at an appreciable rate.

ii. In the second paper the authors discuss the application of the above findings to the culture of *P. knowlesi in vitro.* Two different techniques have been perfected—one, which is termed the rocker-dilution technique consists in diluting parasitized blood with a nutrient medium in a special cylindrical vessel which is provided with three chimneys—a large central one through which parasitized blood and medium can be introduced and two smaller ones near the ends for the maintenance of the gas phase consisting of a 5 per cent. CO_2–95 per cent. air mixture. Into the vessel are introduced 6 cc. of a mixture of medium, parasitized blood and normal blood, and during incubation it is rocked. In the rocker perfusion technique two methods have been employed, in the one medium is passed continuously through a coiled Cellophane tube which is immersed in a quantity of the blood mixture, while in the other the blood mixture in a tube covered at one end with a Cellophane membrane is immersed in a bath of medium. The various techniques are described in detail with the aid of diagrams.

iii. In the third paper an account is given of the results of the culture experiments. It has been possible to obtain normal growth, segmentation and re-invasion in 24 hours in a medium containing only substances of known structure and purity. To obtain subcultures, however, a small amount of normal blood or serum must be added. Subcultures have been obtained for seven generations, the infectivity of the final generation being demonstration by inoculation into normal monkeys. The effects of changes in the medium and in the gas phase have been carefully studied. A high percentage of O_2 inhibits multiplication, but normal development takes place under a gas phase approximating that of alveolar air. A lower percentage of O_2 (0·37 per cent.) is equally satisfactory. Growth is better in normal plasma than in plasma from a parasitized monkey. The inhibitory effect of the latter is removed by heating to 56°C. for 30 minutes. It has been shown that the parasites used glucose and *p*-aminobenzoic acid, while absence of blocks of nutrient such as purines and pyramidines, amino acids or water-soluble vitamins, has been shown to have a deleterious effect on growth. The assay of the nutritional requirements of the parasites is hampered by the nutrient arising from the red cells and plasma which are necessarily present. However, the successful cultivation of parasites in cells washed free of plasma and resuspended in synthetic medium containing 1 per cent. serum albumin seems to be a step towards removal of these interfering factors.

The three papers noted above review the previous work which has been carried out by other observers on the cultivation of malarial parasites, and on their nutritional requirements. They give in detail the methods employed in

this investigation, the composition of the various media tested and the results obtained. Those interested in the biochemical aspects of the subject should study the papers themselves. *C. M. Wenyon.*

SHEN, S. C., FLEMING, Eleanor M. & CASTLE, W. B. **Osmotic and Mechanical Fragilities of Erythrocytes of Monkeys infected with *P. knowlesi* Malaria.** *Proc. Soc. Exper. Biol. & Med.* 1946, Nov., v. 63, No. 2, 419–22, 1 fig.

"Experiments with the blood of control and of *Macaca mulatta* monkeys infected with *Plasmodium knowlesi* indicate that the parasitized red blood cells develop increased osmotic fragility and *in vitro* are selectively destroyed by mechanical trauma. Consequently, it is suggested that because of this increased mechanical fragility of the red blood cells the motion of the circulation *in vivo* plays a part in the production of the hemolytic anemia in malaria of monkeys and, by analogy, of man."

HAAS, V. H., WILCOX, Aimee and EWING, Frances M. **Non-Pigmented Forms of *Plasmodium gallinaceum* in Chick Embryos : Water Color Plates.** *J. National Malaria Soc.* 1947, June, v. 6, No. 2, 122–3, 31 coloured figs. on 2 pls.

Colour plates illustrating the paper abstracted in this *Bulletin*, 1946, v. 43, 1114.

DOWNS, W. G. **Infections of Chicks with Single Parasites of *Plasmodium gallinaceum* Brumpt.** *Amer. J. Hyg.* 1947, July, v. 46, No. 1, 41–4.

Single parasites were isolated with a Chambers micromanipulator from infected chicken blood, diluted 1 in 2,000 with a mixture of 2 parts of 2 per cent. sodium citrate in normal saline and 1 part of normal chicken plasma. The material was inoculated into the hearts of chicks less than 10 days old. Four out of 46 chicks became infected and the disease ran the normal course, except for a prolongation of the incubation period and the greater synchronicity of schizogony. Gametocytes of both sexes were observed. Subinoculations were made, and in the new birds the usual exoerythrocytic cycle was demonstrated. Thus, a single trophozoite of *P. gallinaceum* has potentialities for the initiation of schizogonous, gametogonous and exoerythrocytic cycles.

P. C. C. Garnham.

BALL, G. H. **Attempts to cultivate the Mosquito Phase of *Plasmodium relictum*.** *Amer. J. Trop. Med.* 1947, May, v. 27, No. 3, 301–7, 4 figs. [15 refs.]

Culex tarsalis mosquitoes were infected with *Plasmodium relictum*. After six days, the insects were kept at a constant temperature of 18°C. for 40 or 50 hours and were deprived of food and water. After this interval the majority of stomachs became free of bacterial and fungal contaminants. Then, with aseptic precautions, the mid-gut was dissected out and placed in the culture medium in a Carrel flask. Several media were employed, Trager's medium, Trager's medium plus extra glucose, and different types of synthetic media (Kidder and Dewey). Various mixtures of gases were introduced into the flasks and the correct *p*H was maintained by gassing the medium daily with a mixture of sterile CO_2 and free air.

The stomachs remained alive for as long as 10 days, as proved by contractibility and by the normal structure of the nuclei, but the oöcysts remained cytologically completely unchanged, and no development occurred. This is apparently the first recorded attempt to cultivate *in vitro* the sporogonic stages of *Plasmodium* [it failed possibly owing to asphyxiation of host tissue or of the oöcysts themselves.] *P. C. C. Garnham.*

STAUBER, L. A. & WALKER, H. A. **Preparation and Properties of Erythrocyte-free Avian Plasmodia.** *Proc. Soc. Exper. Biol. & Med.* 1946, Nov., v. 63, No. 2, 223–7, 4 figs. on 1 pl. [11 refs.]

In order to prepare avian malarial parasites for use as an antigen, it is necessary that the parasites should be completely freed from the erythrocytes originally containing them. With nucleated blood corpuscles, this is not as simple as it is with non-nucleated, but the authors have devised a satisfactory technique. They first relieve the cells of their plasma membranes with saponin and then add an enzyme which destroys the nuclei, though leaving intact (but not alive) the parasites and incidentally the leucocytes. Nineteen parts of duck blood infected with *P. lophurae* are drawn into 1 part of 0·9 per cent. saline, buffered at *p*H 7·0 and containing 300 mgm. per cent. of heparin. Centrifugation at 2,000 for 10 minutes brings down the cells, which are washed with 2–3 volumes of buffered saline. The cells are then suspended in 20 times their volume of 1–5,000 saponin in buffered saline, centrifuged for 20 minutes and the material is subsequently suspended in 10 times its volume in Seitz-filtered enzyme solution prepared by dissolving 100 mgm. of trypsin and steapsin in 100 cc. of saline buffered at *p*H 5·0–5·5. Immediate centrifugation is done and the material is again suspended in 10 times its volume of enzyme solution, with occasional shaking for 1½ hours. After washing twice with buffered saline, the suspension is finally made up to a total volume of 10 ml., in buffered saline containing 0·2 per cent. formalin. *P. C. C. Garnham.*

TRAGER, W. **The Development of the Malaria Parasite** *Plasmodium lophurae* **in Red Blood Cell Suspensions in Vitro.** *J. Parasitology.* 1947, Aug., v. 33, No. 4, 345–50. [11 refs.]

The author employs the complicated medium devised in recent years by American malariologists [TRAGER, BALL, ANFINSEN, GEIMAN, and others: see this *Bulletin*, 1942, v. 39, 440; *ibid.*, 1943, v. 40, 675; *ibid.*, 1945, v. 42, 867] for growing malarial parasites. Three extracts are prepared: (1) The culture medium, containing essentially an excess of glucose and calcium pantothenate; (2) a red cell extract; and (3) the parasitized red blood cell suspension with a relatively low parasite density. The cultures are incubated at 40°C. in Erlenmeyer flasks through which moist 95 per cent. air–5 per cent. CO_2 is passed, and they are placed on a rocking machine. Subculture is performed after two days.

Regular multiplication occurs for two days, after which the parasites degenerate. Such cultures are really tissue cultures, rather than ordinary bacterial cultures in non-living media; they differ from the growths of the older methods in that true multiplication of the parasite occurs and studies on its physiology are made possible. For instance, it has been shown that the development of *P. lophurae* is hampered by a high oxygen tension and is favoured by glucose and calcium pantothenate.

[The composition of the culture medium is so complex that it is unlikely that the method will come into routine use.] *P. C. C. Garnham.*

HUFF, C. G., COULSTON, F., LAIRD, R. L. & PORTER, R. J. **Pre-Erythrocytic Development of** *Plasmodium lophurae* **in various Hosts.** *J. Infect. Dis.* 1947, July–Aug., v. 81, No. 1, 7–13, 17 coloured figs. on 1 pl.

This paper is a continuation of the authors' studies on the development of the sporozoite of the different species of avian malaria. The cycle has already been worked out for *P. gallinaceum*, *P. cathemerium* and *P. relictum*; here *P.*

lophurae is studied. Turkeys, ducks, chickens and guinea-fowl were inoculated into the wing skin with sporozoites from infected *Aëdes albopictus*. Biopsy at the site was then performed at varying intervals.

Normal developmental forms were almost exactly similar to *P. gallinaceum*, but a large number of degenerating parasites were encountered, more particularly in the duck and least often in the guinea-fowl.

The discussion in this paper raises a number of points of considerable interest and should be read in full. The authors state that as a result of these studies, it is no longer possible to measure susceptibility in the host solely by the degree of parasitaemia. Some parasites, *e.g. P. lophurae* in the guinea-fowl, exhibit numerous pre-erythrocytic forms, but a mild blood infection. The suitability of a host for a given parasite may be measured by (1) the ease by which it is adapted in the blood passage; (2) its infectiousness to mosquitoes; (3) the ability to produce exo-erythrocytic stages; and (4) the number and normality of the cryptozoites and metacryptozoites.

It is suggested that the pre-erythrocytic stages of malarial parasites will fall into the following groups [the species marked with an asterisk are those in which this stage is known]:—

1. Parasites with exo-erythrocytic stages of *P. gallinaceum* type: *P. gallinaceum*,* *P. lophurae**, *P. cathemerium**, *P. relictum**, *P. durae*, *P. circumflexum* and possibly *P. mexicanum* (which includes both *gallinaceum* and *elongatum* types in its exo-erythrocytic stage).

2. Parasites with exo-erythrocytic stages of *elongatum* type: *P. elongatum*, and perhaps *P. mexicanum*.

3. A doubtful category, where exo-erythrocytic forms are at present unknown. This included other species of avian and saurian malaria and monkey, bat and human malaria. *P. C. C. Garnham.*

BLACKWATER FEVER.

LALICH, J. J. **The Influence of Injections of Homologous Hemoglobin on the Kidneys of Normal and Dehydrated Animals.** *J. Exper. Med.* 1947, Aug. 1, v. 86, No. 2, 153–8, 3 figs. on 1 pl.

The author investigated the effects of intraperitoneal injections of homologous haemoglobin in normal rats and guineapigs, and of intravenous injections in rabbits. The haemoglobin solution used was prepared from homologous heart blood laked in distilled water; stroma was removed by centrifuging. No renal lesions were observed in the rats, most of which developed haemoglobinuria, or in the guineapigs. "Dehydration" was produced in rabbits by withholding water for 1 to 5 days. Blood non-protein nitrogen was raised in 8; three died in uraemia. Pigment casts, dilated tubules, and necrosis of tubular epithelium were observed in the animals deprived of water for the longest periods, and one or more of these features appeared in the other animals. There were apparently no control experiments in normal rabbits. The author claims that haemoglobinuric nephrosis "can be produced consistently in rabbits when dehydration precedes the intravenous injections of hemoglobin," and suggests that similar studies should be made in other species. *B. G. Maegraith.*

LUCKÉ, B. **Lower Nephron Nephrosis. (The Renal Lesions of the Crush Syndrome, of Burns, Transfusions, and other Conditions affecting the Lower Segments of the Nephrons.)** *Milit. Surgeon.* 1946, Nov., v. 99, No. 5, 371–96, 16 figs. (12 on 3 coloured pls.) [92 refs.]

This is an excellent account of the renal lesions seen in blackwater fever, crush injury, incompatible transfusion and other conditions, usually referred to in Britain as renal anoxia. The pathological picture and pathogenesis are clearly discussed.

The author states that the syndrome was the most frequent form of renal dysfunction met in members of the United States Army during the recent war, and the account is based on records and material from over 500 cases. The syndrome appeared in crushing injury, non-traumatic muscular ischaemia, eclampsia, burns, incompatible transfusion, blackwater fever, other intravascular haemolyses, heat-stroke, sulphonamide and other forms of poisoning. The essential lesions in the kidney were restricted to the lower segments of the nephrons and comprised focal degeneration or necrosis, " heme casts, secondary inflammatory reactions in the surrounding stroma, and thrombosis of thin walled veins." The pathogenesis could not be established. Factors concerned were degradation products of haemoglobin and myoglobin, physicochemical alteration of body fluids and blood, shock and disturbances of renal flow arising from ischaemia of the kidney. Renal insufficiency, according to the author, arose from the activity of three factors " mechanical blockage of the tubular lumen by casts, inadequate glomerular filtration, and disturbances of electrolyte balance of body fluids."

The original paper is recommended to all interested in this subject.

B. G. Maegraith.

TRYPANOSOMIASIS.

VAN HOOF, L. M. J. J. **Observations on Trypanosomiasis in the Belgian Congo.** *Trans. Roy. Soc. Trop. Med. & Hyg.* 1947, July, v. 40, No. 6, 728–54. [30 refs.] Discussion 755–61 [CHESTERMAN, C. C.; DAVEY, D. G.; HOARE, C. A.; LOURIE, E. M.; VAN HOOF, L. M. J. J. (in reply)].

This is the second Royal Society of Tropical Medicine and Hygiene Chadwick Lecture.

For many years past, the campaign against sleeping sickness in the Belgian Congo has depended mainly on mass treatment of the affected population. Each year since 1926 between about 2 million and 5 million people have been examined, those found to be infected being subjected to courses of treatment, but instead of an ever-diminishing incidence of new infections, the original over-all rates of about 1 to 1·2 per cent. led to an equilibrium, maintained for the past decade, at about 0·25 per cent. Coincidentally, from practically all endemic zones, came reports of an increasing prevalence of cases resistant to arsenical treatment. Before 1938 about 7 per cent. of new cases in the Léopoldville area, for example, were tryparsamide-resistant. The proportion has since reached 50 per cent. or more in this as well as in a number of other areas. So serious has the incidence of arsenic-resistance become that since 1938 the Directorate of Medical Services has believed it necessary to modify the programme of mass therapy, and to give greater prominence to chemoprophylaxis by suramin and diamidines. This trend was accelerated because of staff shortages during the war. In 1946, there were 50,000 subjects under observation after protection with pentamidine and propamidine, and there should be a total of 175,000 in 1947. A single dose of pentamidine is believed to confer protection

for 6 months, as compared with only 3 months in the case of suramin [VAN HOOF, HENRARD and PEEL, this *Bulletin*, 1947, v. 44, 979]. Propamidine has been found to exercise about the same degree of protection as pentamidine, and detailed reports will appear later, but meanwhile it is noted that pentamidine is preferred, since a number of unexpected toxic effects, such as abortion, have been encountered in the use of propamidine.

The increasing prevalence of trypanosome strains resistant to arsenicals may be attributable either to an actual production of such strains as a result of treatment, or, as suggested by BARLOVATZ [this *Bulletin*, 1933, v. 30, 776], to a suppression of the naturally sensitive strains, allowing those which are naturally resistant to predominate. VAN HOOF believes that the available evidence points towards the latter mechanism as being more responsible than the former for the great increase of arsenic-resistance in the Belgian Congo. The idea that naturally resistant strains may occur has frequently appeared in the literature, since attention was drawn to this feature during early trials of atoxyl by BRODEN and RODHAIN about the year 1905. Actual proof of the occurrence of such naturally resistant strains is, however, difficult in the Belgian Congo, where arsenicals are widely employed for so many diseases, but an appreciable incidence of resistant cases is frequently encountered in areas where the influence of any previous arsenical therapy may be reckoned as negligible.

With regard to the development of resistance during treatment, trypanosomes from patients who have relapsed after standard courses of tryparsamide treatment do not necessarily exhibit any resistance. One patient was observed for 15 years, during which he relapsed repeatedly in spite of repeated tryparsamide treatment. Trypanosomes examined at the fourth relapse still failed to show evidence of resistance. In experimental animals, the facility with which resistance arises during treatment is extremely variable under conditions which are kept fairly constant. The protocols reproduced to illustrate these conclusions are, however, not easy to interpret unequivocally.

VAN HOOF says that he has modified his earlier opinion on the cyclical transmissibility of acquired resistance, by now concluding that this character is not invariably transmitted by *Glossina*. The hypothesis is advanced that "a resistant strain is generally composed of trypanosomes with varying degrees of resistance; the more resistant constituting the minority which assures the survival of parasites after administration of a trypanocidal drug. When this strain is transmitted mechanically—a method which uses a large number of flagellates in a few drops of blood—the resistant character remains constant in the succession of animals inoculated. When, on the other hand, it is transmitted by the fly, two factors intervene to bring about a selection between resistant trypanosomes and others: the small number of flagellates necessary for a cycle, and the fact that the ability to develop in the fly diminishes in trypanosomes which have been affected by trypanocidal drugs while at the same time their resistance increases".

The question whether trypanosomes in the deep organs present the same reactions to arsenicals as those in the peripheral blood has been examined by studying trypanosomes taken from the cerebrospinal fluid. In general, such trypanosomes reacted to arsenicals in the same way as those taken from the blood. However, they were only occasionally transmissible by tsetse, and usually could not be grown in the medium of BRUTSAERT and HENRARD [this *Bulletin*, 1938, v. 35, 704].

As for the treatment of advanced cases of tryparsamide-resistant sleeping sickness, VAN HOOF has been very favourably impressed with melarsen oxide [WEINMAN and FRANZ, this *Bulletin*, 1946, v. 43, 207]. Melarsen [FRIEDHEIM, this *Bulletin*, 1941, v. 38, 634] is too toxic and is not recommended.

Just as there are probably trypanosome strains naturally resistant to arsenicals, so there is evidence of strains naturally resistant to tartar emetic and to suramin, respectively. Strains resistant to these compounds are, however, not as common as those resistant to arsenicals. They occur especially in areas where there are many arsenic-resistant cases.

The possible rôle of domestic animals as a reservoir of *T. gambiense* is not altogether negligible. Natural infection of a dog has been observed. The parasite can be maintained by cyclical transmission in pigs for many years [VAN HOOF, HENRARD and PEEL, this *Bulletin*, 1943, v. 40, 367; 1944, v. 41, 466]. Trypanosomes are remarkably scanty in the blood of pigs, but transmissibility is high in the first passages, decreasing towards the end of a series of infections. The situation appears to be similar in goats. Trypanosomes harboured by these various species of domestic animal (or by antelope) remain cyclically infective for man, though this property may disappear after a prolonged stay in any given host.

Variations in virulence for man are described, within three categories [LESTER, this *Bulletin*, 1934, v. 31, 569; VAN HOOF, HENRARD and PEEL, this *Bulletin*, 1938, v. 35, 698]. The most virulent of these three types occurs sporadically in all areas and presents many of the features of infection by *T. rhodesiense*, including resistance to tryparsamide. The trypanosomes from such cases, especially in relapses after treatment, are only feebly, or not at all, capable of fly transmission.

T. brucei is rare in the endemic sleeping sickness areas, corresponding with the paucity of game. Experiments with this trypanosome are described. The work was done with three strains, obtained from natural infections in a dog, a pig and a goat, respectively. Fly-transmitted infections of these strains were practically symptomless in pigs, tending to die out in 12 to 15 months; serious in goats, with brain lesions, keratitis and c.s.f. changes, ending fatally in 3 to 5 months; and of the classically acute type in dogs. The monkey *Cercocebus galeritus agilis* becomes infected only with difficulty. Seven human volunteers proved refractory to infection by the bite of tsetse flies infected even with serum-resistant trypanosomes, though one of the subjects showed trypanosomes in the blood transiently, about 3 weeks after being bitten by a single infected fly. Thereafter his blood became permanently free of trypanosomes, and he never developed any symptoms.

Immunity in man and animals was found to be only of short [unspecified] duration in man and animals cured of *T. gambiense* infections. Unpublished work by BRUTSAERT and HENRARD showed that the blood of an untreated patient inhibits cultures of *T. gambiense*, but no such inhibition is exercised by blood from a cured or apparently cured patient. The blood of an animal carrier of *T. rhodesiense* or *T. brucei* prevents the growth of *T. gambiense* and inversely, but *T. congolense* grows in the presence of blood from carriers of any of the other trypanosomes mentioned.

As for variations in the cyclical transmissibility index, both in man and in animals the index is at a maximum at the height of infection, that is, in the earliest stages of infection, and it diminishes to nil as the subject passes into the chronic stage. It is not possible to forecast the degree of transmissibility merely by the number of trypanosomes in the blood at the time of feeding tsetse, without due regard to the host species concerned, since the index may be very high in a pig even when diagnosis can be made only by culture, and it may be nil in heavily infected guineapigs. In any individual man or animal, however, the index varies directly with the intensity of blood infection. It is diminished by chronicity of infection, by drug resistance, and by exalted virulence. No evidence was obtained that transmissibility is a function mainly of short forms of the trypanosome, as had been postulated by M. ROBERTSON

[*Report of the Sleeping Sickness Commission*, 1912, 12, 138; *ibid.*, 1913, 13, 94], or of any other morphological type.

In the discussion following this lecture, Dr. C. C. CHESTERMAN drew special attention to the remarkable scale on which mass treatment has been conducted in the Belgian Congo. About 10 years ago, nearly half the entire population was examined annually, a feat probably unequalled in any other territory. He also mentioned that for the past 50 years, 100,000 people have been dying annually from sleeping sickness in Central Africa, but the number has been greatly reduced. This improvement, together with advances in other directions, is likely to lead eventually to serious population pressure, as has developed in India. Dr. D. G. DAVEY referred to the danger of producing cryptic infections by the use of prophylactic treatment. Dr. C. A. HOARE pointed out that BRUTSAERT and HENRARD's *in vitro* demonstration, noted above, of immunological relationships between *T. gambiense*, *T. rhodesiense* and *T. brucei* lends further support to the close affinity between these trypanosomes already established on morphological grounds. The present reviewer discussed aspects of the increased prevalence of arsenic-resistance and the incidence of resistance to tartar emetic in the Belgian Congo. He thought that the evidence thus far produced did not yet justify the conclusion that melarsen oxide is valuable in the treatment of advanced sleeping sickness. *E. M. Lourie.*

WEINMAN, D. **Cultivation of African Sleeping Sickness Trypanosomes on Improved, Simple, Cell-free Medium.** *Proc. Soc. Exper. Biol. & Med.* 1946, Nov., v. 63, No. 2, 456–8.

The author has produced a new solid medium for the cultivation of *T. gambiense* and *T. rhodesiense*, of the following composition: (1) Base (autoclaved portion):—nutrient agar 31 gm., distilled water, to make 1 litre; (2) Non-autoclaved portion:—inactivated human citrated plasma 12·5 cc., human red cells 12·5 cc. To make 100 cc. of medium 25 cc. of (2) are added to 75 cc. of the base (1). The medium is put up in Kolle flasks or slanted in test-tubes, which are kept at room temperature after inoculation of the trypanosomes. In this medium growth is abundant, the time of development depending on the size of the inoculum. When the inoculated blood contains from 1,000 to 10,000 trypanosomes growth in culture is detectable in 5 days and is heavy after 10; when only 10–100 trypanosomes are present the cultures are positive after 10 days. Heavy growth is usually maintained for 10 days, after which the number of trypanosomes declines, being scanty after 35–40 days, though sometimes persisting for more than 60 days. The maintenance of sub-cultures may be achieved by freezing the cultures and storing them for prolonged periods at −70°C. [see WEINMAN & MCALLISTER, below, p. 1,100]. One flask culture may yield from 10 to 15 million trypanosomes, which can be washed in Tyrode's solution and concentrated by centrifugation. Multiplication in culture is intense, an inoculum of 100 trypanosomes giving rise to about 30 million in a few weeks. *C. A. Hoare.*

FAIRBAIRN, H. & CULWICK, A. T. **The Modification of *Trypanosoma rhodesiense* on Prolonged Syringe Passage.** *Ann. Trop. Med. & Parasit.* 1947, May, v. 41, No. 1, 26–9.

A comparison is made of two substrains of a strain of *T. rhodesiense* isolated from man twelve years previously. One substrain was maintained in Tanganyika in serial passages through sheep by cyclically infected tsetse flies, and the other substrain was kept in England, where it was maintained by syringe passages through mice. While the African substrain was polymorphic, being

represented by long, intermediate and short forms, negatively or positively charged [see this *Bulletin*, 1947, v. 44, 649], the English substrain had become monomorphic and was represented only by long forms. Furthermore, their mean length was reduced to 23·29μ, as compared with 28·63μ of the corresponding forms in the cyclically transmitted substrain. Since syringe-passaged strains of trypanosomes of the *brucei* group gradually become more sensitive to arsenicals, the results of tests of such drugs upon laboratory strains cannot be applied to normal cyclically transmitted strains of man in Africa. Hence the authors conclude " that, if chemotherapeutic research in trypanosomiasis is to be really effective, all new drugs should be tested on strains of *T. rhodesiense* which are polymorphic and which have been, and are being, transmitted by *Glossina*." *C. A. Hoare.*

FERREIRA, F. S. da C. Sobre a biologia da *Glossina palpalis* da Guiné Portuguesa. I. Nota preliminar. [**The Biology of *G. palpalis* in Portuguese Guinea.**] *An. Inst. Med. Trop.* Lisbon. 1946, Dec., v. 3, 93–141, 1 text fig. & 12 figs. on 6 pls. [31 refs.] English summary.

Although the Portuguese colonies in Africa are infested by tsetse and affected by trypanosomes, less work has been done there than in certain other parts of the Continent. One therefore welcomes this paper which is an account of work on *Glossina palpalis* carried out in Portuguese Guinea. In this territory *G. palpalis* is much the commonest species of tsetse. The information here about the biology of the fly is interesting as confirming investigations carried on in other territories, but it does not contain a great deal that is new.

Investigations of the effects of climatic factors on the physiology of the fly are given, with some notes on trapping and other control measures. Epidemiological investigations suggest that there has been an increase in the proportion of flies infested with polymorphic trypanosomes within the last 15 years in Portuguese Guinea. *Kenneth Mellanby.*

PINTO, A. R. El diagnóstico de laboratorio de la enfermedad del sueño y su relación con la revisión de enfermos. [**The Laboratory Diagnosis of Sleeping Sickness and its relation to Re-examination of Patients.**] *Med. Colonial.* Madrid. 1947, Aug. 1, v. 10, No. 2, 88–100.

VAN DEN BERGHE, L. Trypanosomose d'un poussin éclos après inoculation chorio-allantoidienne de *Trypanosoma evansi.* [**Trypanosomiasis in a Chick hatched after Inoculation into the Chorio-Allantoic Membrane with *Trypanosoma evansi.***] *Acta Biol. Belgica.* 1941, v. 1, No. 1, 146–51, 1 chart.

The subject-matter of this paper has been more fully reported in a later publication: see this *Bulletin*, 1945, v. 42, 871.

MOULDER, J. W. **Effect of Age of Infection upon the Oxidative Metabolism of *Trypanosoma lewisi*.** *Science.* 1947, Aug. 22, 168–9.

SCHUELER, F. W., CHEN, G. & GEILING, E. M. K. **The Mechanism of Drug Resistance in Trypanosomes. I. A Method for differentiating Strains of Resistant Trypanosomes.** *J. Infect. Dis.* 1947, July–Aug., v. 81, No. 1, 14–18, 2 figs.

A method has already been described by CHEN and GEILING [this *Bulletin*, 1946, v. 43, 315] for measuring the antitrypanosome effect of antimonials, by determining the extent to which these compounds inhibit the parasites' utilization of glucose *in vitro*.

The method has now been used for measuring the antitrypanosome effect of an arsenical (the arsenoxide " Mapharsen ") on three strains of *T. equiperdum*, one of which is normally sensitive, one slightly resistant, and the other highly resistant to arsenicals ; in this way the strains were compared with one another quantitatively in respect of the degree of resistance exhibited by each. The slightly resistant strain had been obtained from the sensitive one by treating infected rats with small doses, and also by *in vitro* exposure of the trypanosomes to non-sterilizing concentrations of the arsenoxide. The manner in which resistance arose in the third strain is not stated.

Where the anti-trypanosome effect was measured by determining the concentration of arsenoxide necessary to effect a 50 per cent. reduction in glucose utilization under the experimental conditions described, the relative degrees of resistance of the three strains were found to be in the proportions of 1, 2 and 7. Appropriate treatment of the data concerned, showed the differences to be statistically significant.

It is arguable to what extent inhibition of glucose utilization reflects the lethal action of the drug on the parasite. It is suggested that 15 per cent. inhibition might be suitable as a measure of mere toxicity, and 90 per cent. as representative of lethal action. In the former case, the differences between the resistant factors of the strains are considerably less, and in the latter considerably greater, than in the proportions of 1 : 2 : 7 obtained when 50 per cent. inhibition is the criterion.

The strain of low resistance lost this character completely in the course of 4 serial passages in rats. In the highly resistant strain, however, the resistance seems to be permanent. It is therefore concluded that there must be fundamental differences in the nature of the resistant properties exhibited by these two strains.

E. M. Lourie.

BARLOW, F. & HADAWAY, A. B. **Preliminary Notes on the Loss of DDT and Gammexane by Absorption.** *Bull. Entom. Res.* 1947, Aug., v. 38, Pt. 2, 335–46.

These experiments were done in connexion with tsetse fly control and, accordingly, *Glossina palpalis*, as well as mosquitoes, were used to give biological assessment of insecticidal action. Other data were obtained by chemical estimations.

Tests with sprayed vegetation.—Two types of leaves, both common near Lake Victoria, were used, one with a hard surface, and one a soft and hairy one. Leaves on growing plants were sprayed with DDT or *gamma* benzene hexachloride (" gamma BHC ") in kerosene-cotton-oil solution or in emulsions, and examined at intervals, chemically and biologically. By comparing external leachings with extractions from dried and macerated leaves, it was found that from 20 to 50 per cent. of the insecticide was absorbed into the interior of the leaves. The amounts of insecticide both internal and external, progressively declined with ageing.

Applications to mud and thatch.—These tests were made in connexion with mosquito control, but *Glossina* was used in most of the tests. Applications of DDT and *gamma* BHC were made to dried mud, thatch and to glass plates. It was found that applications of solutions or emulsions of insecticides to dried mud resulted in considerable penetration of the insecticide below the surface ; with aqueous suspensions, on the other hand, the insecticide was deposited superficially. These results of chemical estimations of different levels were supported by biological data which showed that the aqueous suspension deposits were more highly insecticidal than that of solutions or emulsions.

The same effects were observed *to a lesser extent* with grass thatch and not at all with the impermeable glass plates.

Applications to limewash.—DDT sprayed in solution on to glass plates coated with a thin film of limewash was rendered much less insecticidal than lower deposits applied to plain glass. Chemical extractions proved that the DDT was not altered chemically to any great extent; apparently it was physically absorbed or merely masked by the particles of lime. Experiments showed that this effect was much less marked if the DDT were applied in suspensions (containing only 50 per cent. inactive material). Results with a suspension containing a "sticker" (sulphite lye) showed that this material was also liable to mask DDT toxicity, presumably by coating the particles.

J. R. Busvine.

See also p. 1103, HACKMAN, **The Persistence of DDT on Cattle.**

HAWKING, F. **Growth of Protozoa in Tissue Culture. III.** *Trypanosoma cruzi.* *Trans. Roy. Soc. Trop. Med. & Hyg.* 1946, Dec., v. 40, No. 3, 345–9, 13 figs. (2 on pl.).

The author employs his *P. gallinaceum* technique [this *Bulletin*, 1946, v. 43, 410] for growing *T. cruzi* in tissue cultures of heart, liver and subcutaneous tissues of rat embryos and of chick embryo brain. The trypanosomes were obtained free of red blood cells by centrifugation of infected mouse blood and were introduced into the growing implants on the third day. They multiplied extra-cellularly for two days, then became fewer until the twelfth day, when free forms had practically disappeared, to reappear later in small numbers up to the 59th day. After the sixth day, developmental forms were numerous in cardiac muscle cells, and in reticulo-endothelial cells. In many cases, the parasitized cells appeared to be damaged—the cytoplasm was vacuolated, and the nucleus pyknotic. The leishmanoid forms sometimes showed two parabasal bodies, whilst later a tiny granule (? blepharoplast) appeared beside this structure. Eventually the complete trypanosome is formed and becomes extra-cellular.

P. C. C. Garnham.

FLOCH, H. & DE LAJUDIE, P. Recherches sur la trypanosomiase humaine américaine en Guyane française: *Rhodnius prolixus* et *Rhodnius pictipes* vecteurs naturels de choix de *S. cruzi.* [*R. prolixus* **and** *R. pictipes* **as Natural Vectors of** *T. cruzi* **in French Guiana.**] *Bull. Soc. Path. Exot.* 1947, v. 40, Nos. 5/6, 157–60.

HAUSCHKA, T., SAXE, L. H., Jr. & BLAIR, Margaret. *Trypanosoma cruzi* **in Treatment of Mouse Tumors.** *J. National Cancer Inst.* 1947, Feb., v. 7, 189. [Summary taken from *J. Amer. Med. Ass.* 1947, Sept. 6, v. 135, No. 1, 60.]

Hauschka and his co-workers observed that infections with Trypanosoma cruzi (Brazil strain) significantly retarded the growth of squamous epithelial carcinoma 119 in A mice, mammary adenocarcinoma in C3H mice and sarcoma 37 in white mice of unknown genetic background. Spontaneous breast adenocarcinoma in C3H mice was lightly retarded in growth. The infection was more virulent in A than in C3H mice. The inhibitory effect appears to be nonspecific, since it was frequently accompanied with loss in body weight and parasitemia of vital organs such as heart, lungs and intestine. Cancer cells were rarely parasitized. Leishmanian and crithidial forms of T. cruzi were more abundant in the stroma and the connective tissue surrounding some tumors. Retardation in tumor development did not result in longer survival. Endotoxin prepared from heat killed cultures of T. cruzi (Brazil strain) was

without effect against carcinoma 119 or against transplantable and spontaneous mammary tumors. The authors conclude that the results they obtained are not to be considered as contradicting the results of the Russians, Roskin and Klyueva, as their strain of T. cruzi has not been available to Hauschka and his co-workers.

LEISHMANIASIS.

SEN GUPTA, P. C. **History of Kala-Azar in India.** *Indian Med. Gaz.* 1947, May, v. 82, No. 5, 281–6, 2 maps. [19 refs.]

FENG, L. C. & CHUNG, H. L. **Experiments on the Transmission of Kala-Azar from Dogs to Hamsters by Chinese Sandflies.** *Chinese Med. J.* Peiping Edition. 1941, Nov., v. 60, No. 5, 489–96. [11 refs.]

"1. Transmission experiments of kala-azar from dogs to hamsters by means of sandflies are reported.

"2. Two out of 8 hamsters inoculated intraperitoneally with flagellates from *P. sergenti* var. *mongolensis* fed previously on infected dog developed kala-azar.

"3. Six out of 20 hamsters inoculated intraperitoneally with flagellates from *P. chinensis* fed previously on infected dog develop kala-azar.

"4. One out of 5 hamsters inoculated percutaneously with flagellates from *P. chinensis* also became infected.

"5. Successful transmission of kala-azar from dog to hamsters through the bite of *P. chinensis* is reported for the first time.

"6. These experiments gave further support to the view that *P. chinensis* is the important transmitter of kala-azar in China."

SHIH LU CHANG. **Studies on Haemoflagellates. I. A Semi-Solid Medium and a Fluid Medium with a Solid Base for growing various Species of *Leishmania* and *Trypanosoma cruzi*.** *J. Infect. Dis.* 1947, Mar.–Apr., v. 80, No. 2, 164–71. [10 refs.]

The author has devised two new media for the cultivation of haemoflagellates. One, a semi-solid medium, has the following basic composition : bacto-tryptose, 15·0 gm. ; liver extract powder, 10·0 gm., glucose, 2·0 gm. ; NaCl, 4·0 gm. ; KCl, 0·4 gm. ; Na_3PO_4, 5·0 gm. ; bacto-agar, 2·0 gm. ; distilled water, 1 litre. The medium is autoclaved and 150 ml. horse serum+20 ml. horse haemoglobin solution are added to it. After mixing, the solution is distributed into tubes or flasks. The second medium is fluid, with a solid base ; its ingredients are the same as above, but agar is omitted. The solution is divided into two portions, measuring 600 ml. and 400 ml., respectively. To the larger volume are added 7·2 gm. of agar, the mixture is boiled until the agar is dissolved, and dispensed in 20–25 ml. amounts into Erlenmeyer flasks. These flasks and the 400 ml. of fluid medium are autoclaved, after which the flasks are cooled in an upright position to allow the agar base to solidify at the bottom. To the fluid medium are added 60 ml. horse serum+8 ml. haemoglobin solution and the resulting mixture is dispensed in 15 ml. amounts over the solid base in the flasks. Both media produced much better growth of *L. donovani*, *L. tropica*, *L. brasiliensis* and *T. cruzi* after a shorter period of incubation at room temperature than the

media usually employed for the cultivation of these haemoflagellates. The maximum numbers are reached in about one week in the case of the medium containing the fluid, and several days later in the semi-solid one.

C. A. Hoare.

SHIH LU CHANG & NEGHERBON, W. O. **Studies on Haemoflagellates. II. A Study of the Growth Rates of** *Leishmania donovani, L. brasiliensis, L. tropica,* **and** *Trypanosoma cruzi* **in Culture.** *J. Infect. Dis.* 1947, Mar.–Apr., v. 80, No. 2, 172–84, 7 graphs.

Using the fluid medium with a solid base described in a previous paper (see preceding abstract), the authors studied the rates of growth of cultures of haemoflagellates, the logarithmic rates of growth being expressed by two equations. It was found that *L. tropica, L. brasiliensis,* and *T. cruzi* grew in cultures according to the general pattern of the growth curves for micro-organisms, which have a lag phase, a phase of logarithmic growth, a phase of stationary growth, and a phase of decline. The lag phase was, however, only seen in *L. donovani.* The logarithmic growth phase lasted from two days in *L. tropica* to six days in *T. cruzi.* The growth rate of the different flagellates varied : *L. tropica* was the fastest, followed in order by *L. brasiliensis, T. cruzi* and *L. donovani.* As regards the stationary and declining phases of growth, they varied in cultures of the same species. It was also found that an increase in the temperature of incubation accelerates the growth of *L. tropica* between 17 and 27·5°C., and of *L. brasiliensis* and *T. cruzi* between 17 and 32°C., whereas *L. donovani* showed negative correlation with temperature increase. None of these flagellates grows well at body temperature, *L. donovani* tolerating this temperature least and *T. cruzi* best. The cultures of various species of haemoflagellates can be differentiated macroscopically : *L. brasiliensis* shows a heavy waxy growth which appears like a pellicle on the surface of the liquid ; *L. tropica* shows a streaming turbidity when the culture is agitated ; *L. donovani* has a tendency to grow in large clumps seen in the form of large floccules when the culture is agitated ; *T. cruzi* does not show any marked feature except heavy turbidity and a marked yellowish colour of the culture when this is disturbed.

C. A. Hoare.

DOSTROVSKY, A. & SAGHER, F. **Diagnostic Significance of the Culture Method in Cutaneous Leishmaniasis (Oriental Sore).** *Arch. Dermat. & Syph.* 1946, Nov., v. 54, No. 5, 543–51, 4 figs. [Refs. in footnotes.]

It is noted that in many cases of chronic leishmaniasis of the skin, particularly in relapsing cases, it may be extremely difficult to find leishmania in smears from the lesions or in sections of biopsy material. With a view to assistance in diagnosis, the authors have tested the possibility of obtaining cultures from the lesions in 61 cases. Of these, 26 were early stages of the disease, the leishmania being demonstrated in smears from 12, all but two of which gave positive cultures. Cultures were positive in 8 cases which gave negative smears, while in 6 cases both smears and cultures were negative. Attempts at culture from the scars of healed sores failed on five occasions. Of 30 cases of the late stage of cutaneous leishmaniasis, 18 were negative in smears but positive in cultures, 10 were negative in both and two were positive in both. Some of these were relapsing cases, while others had lesions of several years' duration. It is evident that the culture method of diagnosis is of value, particularly in the old chronic case, in which the lesions may resemble those of lupus or syphilis.

C. M. Wenyon.

FEVERS OF THE TYPHUS GROUP.

GIROUD, P. & JUDE, A. Au sujet de la réaction de fixation du complément. Pouvoir antigène des constituants d'un vaccin antityphique. Résultats chez des vaccinés et des convalescents. [**On the Complement-Fixation Reaction. Antigenic Potency of the Constituents of Anti-Typhus Vaccine. Findings in Vaccinated Persons and Convalescents.**] *Bull. Soc. Path. Exot.* 1947, v. 40, Nos. 5/6, 142–7.

The authors describe tests carried out with antigens prepared from formolized rabbit-lung vaccine. Similar and satisfactory results were obtained from the use of the whole vaccine and also from its rickettsial content and the supernatant fluid obtained by centrifugation.

With very small doses of the whole vaccine as antigen, fixation tests were carried out on the sera of 45 persons who had received the last of three doses of vaccine eight days previously; the reaction is described as very strong in 35 cases; it was strong in four, positive in two, and feeble in one. The remaining three sera were anti-complementary.

The Weil-Felix reaction remained negative in all these cases, whereas there was a rising-titre reaction in every one to the rickettsia-agglutination test.

A curious case is described in which a man, after a heavy bout of drinking, had a sudden rise of temperature to 40·7°C., with diarrhoea and vomiting. Next day his temperature was still high and a Weil-Felix test was carried out. The response was an *OX19* titre of 1–400, an *OX2* titre of 1–800, and an *OXR* titre of 1–100. After the fall to normal of the temperature—about four days later—all the responses were negative except for a titre of 1–50 against *OX19*. Complement-fixation and rickettsia-agglutination tests carried out on both occasions were completely negative.

From all the above findings, the author concludes that the Weil-Felix test is less specific than the other two. *John W. D. Megaw.*

HOLLER, G. & ZAJITSCHEK, R. Eine sehr erfolgreiche, streng kausalpathogenetisch eingestellte Therapie des Fleckfiebers. [**A very Effective and strongly Specific Treatment for Typhus Fever.**] *Med. Klin.* 1944, Apr. 28, v. 40, Nos. 17/18, 247–50, 5 charts.

The authors claim that two new proprietary drugs introduced by the Bayer firm have a powerful antibacterial action on the rickettsiae of typhus fever. The drugs are called "Nitroacridin 3582" and "Rutenol"; the latter is said to be Nitroacridin in combination with arsenic.

The evidence produced to substantiate the authors' claim is a description of five cases treated by one or other of the drugs. One of the patients died; his case is said to have been quoted as a warning against using too small doses of the drugs; another patient had been inoculated with a typhus vaccine; the course of the disease as shown by the charts of the remaining three cases does not appear to differ from that observed in many cases treated without special drugs.

The preparations are claimed to be effective also in trench fever and typhoid fever; if they turn out to be of value, more will be heard of them.

John W. D. Megaw.

FLECK, L. **Specific Antigenic Substances in the Urine of Typhus Patients.** *Texas Reports on Biol. & Med.* 1947, v. 5, No. 2, 168–72.

The author in 1942 injected 10 cc. of urine from a typhus patient into a rabbit by the intravenous route with the result that the animal gave a positive Weil-Felix reaction at a titre of 1–320.

In further experiments it was found that antigens began to appear in the urine of patients on the second day of the fever, and that rabbits inoculated with urine of patients sometimes showed reactions at titres of 1–640 or 1–1,280.

By methods of purification and concentration, which are described in the paper, it was found possible to prepare a protective vaccine from the urine; this vaccine was found protective to guineapigs and was used to vaccinate 500 persons. The after-history of the vaccinated could be obtained only in the cases of the author, his family, and three other persons, all of whom afterwards had mild or abortive attacks of typhus fever, whereas other prisoners in the hands of the Germans at the time had severe attacks with a fatality rate of 30 per cent.

The experiments were carried out while the author was working in the "Ghetto" Hospital at Lwow, Poland, during the German occupation.

John W. D. Megaw.

WEIGL, R. **Immunization against Typhus Fever in Poland during World War II.** *Texas Reports on Biol. & Med.* 1947, v. 5, No. 2, 177–9.

The author expresses his views on the comparative value of the chief typhus vaccines used during the last war.

One million civilians in East Poland were vaccinated with louse vaccine, and although only 25 to 30 louse intestines were used for each person, instead of the full dose of 100 intestines, the effect "exceeded all expectations."

A comparative study in the laboratory showed that the louse vaccine was preferable to the mouse-lung vaccine, of which some batches were entirely inactive.

Strains of *Rickettsia prowazeki* maintained in the author's laboratory by repeated transfer through mice are said to have slowly but steadily approached to the murine type. The author states that vaccines of the murine type do not protect against louse-borne typhus.

Cox-type vaccine, produced early in the war by the Behring firm in Germany, was either valueless or of little protective value, but when prepared in Lwow according to the advice of the author, a much higher standard of potency was reached. Rigorous controls and alternate passages from yolk-sac cultures to lice were employed.

Even the best yolk-sac vaccines were found inferior to the louse vaccine, both in the degree and duration of the resultant immunity.

No details are given of the data on which the above conclusions are based.

John W. D. Megaw.

MOSING, H. **Methods of Evaluation of Typhus Vaccine Potency.** *Texas Reports on Biol. & Med.* 1947, v. 5, No. 2, 173–6, 1 chart.

The author, working at the Weigl Institute in Lwow, found that mice after inoculation with varying doses of louse vaccine responded to the Weigl (rickettsia-agglutination) test at titres which varied directly with the dose of the vaccine. The doses ranged from 0·0025 of a louse intestine to 100 louse intestines, the titres ranged from less than 1–10 to 1–1,280.

The vaccinated mice and controls were challenged by injections of 30 intestines of heavily infected lice. Control mice and mice vaccinated with small doses—up to 0·5 of an intestine—reacted at titres below 1–40 and all of them died. Mice receiving one or two louse intestines reacted at 1–40 and had attacks of typhus from which they recovered. Mice receiving larger doses reacted at higher titres and none of them was attacked.

John W. D. Megaw.

RAYNAL, J. H. Etudes sur le typhus, Son comportement à Chang-Hai de 1938 à 1945. [**Studies of Typhus. The Behaviour of the Disease in Shanghai from 1938 to 1945.**] *Méd. Trop.* Marseilles. 1947, Mar.–Apr., v. 7, No. 2, 91–147, 11 graphs (3 folding). [80 refs.]

This is a more detailed version of a paper already reviewed in this *Bulletin* [1947, v. 44, 893]. It contains the graphs and statistics for which room could not be found in the previous article, and also a full discussion of the relationship between murine and epidemic typhus.

The author claims that the survey carried out from 1938 to 1934 shows :— (1) That murine typhus is constantly present as an enzootic disease in Shanghai ; (2) That human infection of rat or rat-flea origin is liable to occur at any time in sporadic form ; (3) That in exceptional circumstances murine rickettsiae can adapt themselves to transmission by lice from man to man and so give rise to epidemics ; these circumstances include (*a*) the occurrence of epizootics of the disease among rats in the spring season when human lice are specially numerous and active, and (*b*) the existence of crowding and other conditions favourable to louse transmission ; (4) That severe and persistent epidemics cannot occur in Shanghai because louse transmission becomes arrested in very hot weather, so that epidemics such as started in the spring in 1938, 1940 and 1942 in Shanghai come to an end before the murine rickettsiae has become fully adapted to transmission by lice.

An association between rat epizootics and human epidemics is most strongly suggested by the findings obtained in the survey of 1942. The following table shows statistics for that year collated from various parts of the paper :—

	Jan.	Feb.	March	April	May	June
Human cases of typhus fever	34	64	109	199	292	203
Percentage of rats showing *OX19* titres of 1–100 or over	0	16·6	21·8*	7·9	3·4	6·2
Xenopsylla cheopis index among rats	0·17	0·13	0·13	0·04	0·52	1·54

	July	Aug.	Sept.	Oct.	Nov.	Dec.
Human cases of typhus fever	119	37	36	40	39	50
Percentage of rats showing *OX19* titres of 1–100 or over	1·8	12·7	14·1	9·3	17·7	9·1
Xenopsylla cheopis index among rats	4·6	3·3	2·35	2·18	1·65	0·82

*Including one period of 10 days in early March in which the percentage rose to 60.

The author states that two strains of rickettsiae recovered from patients during the 1938 epidemic were of the murine type, though both showed a tendency to approach to the " intermediate " type.

The paper deserves close and critical examination by those interested in the controversial problem of the relationship between murine and epidemic typhus. The number of rats examined in each month is not stated, the total number examined during 1942 was 707, so that the monthly average must have been less than 60 and the average for each 10-day period less than 20.

John W. D. Megaw.

KIRBY, F. G. **Para-Aminobenzoic Acid in the Treatment of Endemic (Murine) Typhus Fever.** *Ann. Western Med. & Surgery.* 1947, July, v. 1, No. 5, 203–5, 1 fig.

" 1. The use of para-aminobenzoic acid and the sodium salt of para-aminobenzoic acid in the treatment of a patient with endemic typhus fever is reported.

" 2. Marked improvement in the general condition of the patient was observed following the oral administration of para-amino-benzoic acid and its sodium salt.

" 3. Toxic effects were not noted following the use of these drugs."

GRIFFITHS, J. T., Jr. **A Further Account of Tsutsugamushi Fever at Sansapor, Dutch New Guinea.** *J. Parasitology.* 1947, Aug., v. 33, No. 4, 367–73, 2 figs.

This paper is a supplement to an important previous article by the author [see this *Bulletin*, 1946, v. 43, 431]. A novel type of rat trap is described; this consists of a 55-gallon oil drum which, after removal of one end, is buried in such a way that the open end remains a few inches above the surface of the ground: wooden paddles are balanced round the edge of the drum and baited so that any rat that approaches the bait falls with the paddle to the bottom of the drum.

The environments studied were (1) climax rain forest, (2) abandoned village and garden areas, and (3) beach and forest margins. There was no evidence that infection had been contracted in the first of these, but the other two were definitely associated with transmission of the disease.

Details are given of the mites found on rats trapped in the area. Most of the rats belonged to the *Rattus concolor* group. The most abundant mite was *Trombicula deliensis*, which was found on rats in all the sites that were surveyed. Larval mites were found attached to the skin of five soldiers working in uncleared rain forest; the mites were *T. wichmanni* (syn. *T. buloloensis*) but no mites of this species were ever found on rats.

Clearing of areas caused no material reduction in the number of rats captured, but the number of mites on each rat was greatly reduced.

In large grassy areas which appeared to be uniform in the matter of risk of infection, there was sometimes a high incidence of the disease among troops occupying one small patch and no cases from other parts of the area.

John W. D. Megaw.

See also p. 1064, ELISHEWITZ, **Epidemic Control with DDT, ANTU and 1080.**

LOUVEAUX, J. Aspects cliniques du " Scrub Typhus " sur le Front Birman. [**Clinical Aspects of " Scrub Typhus " on the Burma Front.**] *Ann. Soc. Belge de Méd. Trop.* 1947, Mar. 31, v. 27, No. 1, 105–38, 5 figs. [23 refs.]

This is a clear and concise description of the signs and symptoms observed in 51 cases of scrub typhus seen during the last four months of 1944 at the Belgian Field Hospital during the advance of the East African Division in North-West Burma.

Nine of the patients died, including four who suddenly became jaundiced 48 hours or less before death.

Only one patient had an eschar; a faint rash was seen in two cases, it was restricted to the back and shoulders. All but two of the patients were East Africans.

John W. D. Megaw.

FINLAND, M. & LESSES, M. F. **Q Fever. Report of a Case.** *New England J. of Med.* 1947, Aug. 21, v. 237, No. 8, 255–8, 2 figs. [18 refs.]

"A case of Q fever occurring in Boston but originating in the vicinity of Naples, Italy, is reported. The clinical features were those of a nonbacterial pneumonia. The diagnosis was suspected on epidemiologic grounds and established by the demonstration of a rise in antibodies for *Rickettsia burneti* in the serum during convalescence."

BÜHLER, F. Zur Ätiologie der Feldnephritis. [**Aetiology of Field Nephritis.**] *Med. Klin.* 1944, June 9, v. 40, Nos. 23/24, 334–7.

The author discusses the many views that have been expressed with regard to the cause of field nephritis. He points out that the disease is by no means new; under the name "war nephritis" it was well known in the 1914–18 war. Special mention is made of an observation made at that time by Töpfer to the effect that lice collected from patients often harboured organisms resembling those of typhus fever and trench fever. Töpfer regarded these organisms as the cause of proliferative changes in the capillary endothelium and damage to the kidneys.

The author agrees with the view of REUTER and SCHÄFER [see below] that the cases seen in the late-spring and summer months differ from those occurring in winter, in that the former group tend to occur as epidemics and are often associated with febrile paroxysms of the trench fever type, and enlargement of the spleen. *John W. D. Megaw.*

SCHÄFER, W. & REUTER, A. Verlaufsformen der Feldnephritis. [**Different Forms of Field Nephritis.**] *Med. Klin.* 1944, June 9 & 23, v. 40, Nos. 23/24 & 25/26, 337–9; 377–81, 4 figs. & 3 charts.

The authors have seen 600 to 800 cases of field nephritis within a year in the northern part of the "Eastern Front" in 1942–43. They were greatly impressed by the great variability of the disease, both in type and in severity.

A detailed description is given of three main types, all of which are regarded as being associated with damage to, and increased permeability of, the whole of the capillary system rather than with special damage to the kidneys.

The commonest type is that in which three cardinal features are present—raised blood pressure, albuminuria, and oedema. The duration was seldom more than one or two weeks; pronounced uraemia was never observed, and there were no deaths. About 40–50 per cent. of the cases belonged to this group.

In the second type, one or two of the above cardinal features were absent and doubt often arose whether these cases could rightly be classified as field nephritis, especially when oedema was the only sign. About 20 per cent. of the cases were of this type.

The most interesting type was that in which there was evidence of an association with trench fever. Although there were great diagnostic difficulties in many of these cases it was believed that about 30 per cent. of the cases fell into this class. The attack of trench fever often preceded the onset of the disease by several weeks.

The authors have already suggested that war nephritis is one of the forms of trench fever [see this *Bulletin*, 1945, v. 42, 204]: in the present study they have obtained evidence that one type of the disease is caused by *Rickettsia quintana.*

[This paper and the one reviewed above are very instructive on the subject of war nephritis, but the impression conveyed by them is that the condition

is a symptom rather than a disease and that it may be a manifestation of various diseases, including undetected infections of different kinds.]

John W. D. Megaw.

YELLOW FEVER.

Fox, J. P. **The Cultivation of Yellow Fever Virus. I. Factors influencing the Multiplication of 17D Virus in Tissue Culture.** *Amer. J. Hyg.* 1947, July, v. 46, No. 1, 1–20, 3 figs. [58 refs.]

Various workers have studied some of the factors which influence the growth of yellow fever virus in tissue culture. Fox has undertaken further experiments in an effort to define these factors more clearly in relation to the growth of the attenuated, culture-adapted 17D strain in cultures of minced 10-day-old chick embryos.

The relation between the concentration of virus in the tissue and in the fluid components was relatively constant over a 4-day period, the concentration in the tissue being approximately 100 times that in the supernatant.

Virus was cultivated for 40 days in one series and for 20 days in a second series of clot-base cultures with the use of a supernatant rich in embryo extract which was partially renewed three times a week.

In studying the type of tissue which would support maximum growth, tissue growth was not observed in cultures of chick-embryo brain, but stained smears suggested that the cells remained viable during the period of cultivation. Both tissue growth and changes in *p*H were observed in cultures of whole chick embryo or embryo without the nervous system. In all instances the maximum tissue growth and the lowest *p*H were reached in 3 days, the greatest proportional change occurring during the first 48 hours. It was during this period that the greatest multiplication of virus usually occurred.

When serum in 50 per cent. concentration is used, tissue proliferation, and thus virus multiplication, are retarded.

Within rather narrow limits, an increase in the amount of tissue resulted in an increase in the amount of virus produced in tissue culture, and above these limits a continued increase in tissue prejudiced virus multiplication.

It has been shown that the stability of yellow fever virus is relatively constant over the usual range of *p*H encountered in tissue cultures.

Maximum titres were essentially the same with all the inocula, but the time at which they were attained varied with the size of the inoculum, being significantly delayed when very small doses of virus were used.

The optimal range of temperature for incubation of cultures was found to be from 32·5 to 35°C.

It was found that cultures could be prepared in advance and stored at 5°C. for at least 11 days prior to inoculation without seriously impairing their usefulness for the cultivation of virus. *F. O. MacCallum.*

Fox, J. P. & Laemmert, H. W., Jr. **The Cultivation of Yellow Fever Virus. II. Observations on the Infection of Developing Chick Embryos.** *Amer. J. Hyg.* 1947, July, v. 46, No. 1, 21–40, 2 figs. [52 refs.]

When inoculated on to the chorio-allantoic membrane of 7-day-old embryos, yellow fever virus multiplied rapidly at the site of inoculation, was found in blood at 12 hours, and at 24 hours in whole embryo, brain and liver with maximum virus concentration on the fourth day.

Virus persisted in the tissues until hatching and was found in the blood of about half the hatched chicks. Antibodies were found 30 days later only in those chicks whose blood had been found to contain virus. Resistance to the virus increased with age at inoculation and embryos 13 days or older when inoculated, hatched nearly as well as uninfected controls.

The principal histological findings were the irregular occurrence of focal necrosis in the muscles and an increase in foci of myelopoiesis.

Virus multiplication in embryos, as in tissue cultures, was related to the dose of virus and the temperature at incubation. Higher concentrations of virus were found in embryos infected when under 10 days old.

Relatively large amounts of virus were required to infect by the allantoic or yolk sac routes or when unmodified virus strains were used.

It was found that development of infection was more closely dependent upon the concentration of virus to which the cells were exposed than upon the number of cells so exposed.

One of the most important observations was that prolonged embryo-to-embryo passage of strain 17D, and the French neurotropic strains, failed to reveal significant changes in character of virus.

As well as the 17D strain, which appeared after many attempts at attenuation of the pantropic virus, PENNA and MOUSSATCHÉ reported in 1939 [this *Bulletin*, 1940, v. 37, 558], that continued embryo passage had resulted in attenuation of the Asibi strain. It is interesting to note that Penna has informed Fox that several attempts to repeat the attenuation of Asibi virus have been unsuccessful. It would seem that the factor governing the production of these valuable variants is still a mystery. *F. O. MacCallum.*

PELTIER. **Control of Anti-Yellow-Fever Vaccine from the Pasteur Institute at Dakar by the Quarantine Commission of U.N.R.R.A. and other Questions relating to Yellow Fever.** *Bull. Office Internat. d'Hyg. Publique.* 1946, Oct.–Nov.–Dec., v. 38, Nos. 10–11–12, 820–23. [French version 816–19.]

See this *Bulletin*, 1946, v. 43, 1135.

CERQUEIRA, N. L. & LANE, J. **Note on *Haemagogus capricornii* Lutz, 1904 (Diptera, Culicidae).** *Proc. Entom. Soc. Washington.* 1945, Dec., v. 47, No. 9, 279–88, 10 figs.

Mosquitoes of the genus *Haemagogus* are important as transmitters of the virus of yellow fever in the forests of Tropical America. The present paper is a contribution to exact systematics and the authors review that subject with particular relation to *H. capricorni*, of which the type locality was near the city of São Paulo, Brazil. They describe with figures the male, female, pupa and larva of a species which occurs in the type locality and which they believe is identical with *H. capricorni* of Lutz 1904. *P. A. Buxton.*

PLAGUE.

ROUSSELOT, R. Les rongeurs de la région de Tehran. [**Rodents of the Teheran Area.**] *Arch. Inst. d'Hessarek.* Teheran. 1947, May, No. 5, 51–61, 7 figs. on 1 pl.

YANG, C. S. **Some Notes and Comments on the "Black Death."** *Chinese Med. J.* Shanghai. 1947, Jan.–Feb., v. 65, Nos. 1/2, 31–6, 1 fig.

LAZARUS, A. S. & GUNNISON, J. B. **The Action of *Pasteurella pestis* Bacteriophage on Strains of *Pasteurella*, *Salmonella*, and *Shigella*.** *J. Bacteriology.* 1947, June, v. 53, No. 6, 705–14. [15 refs.]

Bacteriophage action is not absolutely specific and, as one might expect, may be related to the composition of the bacterial antigenic mosaic. A large batch of *P. pestis* phage was prepared and called the " parent strain ". A further careful purification of this phage provided the " purified phage ".

One of the difficulties of diagnosis of plague, bacteriologically, has been a liability to confuse *P. pestis* with *P. pseudotuberculosis*. In the present study, 27 strains of *P. pseudotuberculosis* were tested for sensitivity to the *P. pestis* phages. Differentiation was possible in most cases, but not to such a degree as to make this test sufficiently reliable for isolation. The problem of specificity was taken further, for these two organisms are rather closely related. It was found, however, that the application of both parent phage, and its *P. pseudotuberculosis* phage adaptation, to *P. septica* was without any lytic action.

Salmonella and *Shigella* strains are not generically related to the *Pasteurella* and yet 3 out of 42 strains of the former and 6 out of 37 of the latter were sensitive to *P. pestis* phage. The plague bacteriophage, in the course of adaptation to Shigella organisms, lost none of its original power to lyse plague bacilli. Phage sensitivity among bacteria has been found to be correlated with the distribution of surface somatic antigens and this finding is supported by these studies. *W. F. Harvey.*

PRINCE, F. M. & WAYSON, N. E. **Addendum to Plague—the Survival of the Infection in Fleas or Hibernating Ground Squirrels.** *Pub. Health Rep.* Wash. 1947, Aug. 8, v. 62, No. 32, 1167–8.

The experiment with ground squirrels (*Citellus richardsonii*) and fleas (*Diamanus montanus*) [see this *Bulletin*, 1947, v. 44, 816] has been repeated with *Citellus townsendii* and the same species of fleas. Experiment No. 1 was set up with 4 squirrels inoculated intracutaneously with *P. pestis* and having 50 normal fleas on each squirrel. After hibernation for 4 months one squirrel developed acute bacteraemic plague 6 days after awakening, while the remaining 3 squirrels and all 42 of the fleas left alive showed no infection. In experiment No. 2 " fifty infected fleas were placed on each of four hibernating squirrels and left with them for 4 months." Four of the 47 fleas that remained alive were infected with plague and one of these placed on a rat produced plague buboes in the latter. Thus it may be concluded " that a squirrel in whose skin plague is introduced while the animal is in hibernation in October may awaken 4 months later and subsequently develop acute manifestations of the local lesion and a lethal bacteremia. Also, that a flea infected in October can retain the infection and transmit it during the following March." *W. F. Harvey.*

ELISHEWITZ, H. **Epidemic Control with DDT, ANTU and 1080.** *Soap.* New York. 1947, May & June, v. 23, Nos. 5 & 6, 127–9, 151 ; 153–9, 193. [18 refs.]

Detailed instructions are given for controlling epidemics of plague or murine typhus by attacking both the insect vector and the rodent reservoir. The fleas are destroyed first, before the rats, to prevent their migration from dead rats to seek human hosts. Two, 5 or 10 per cent. DDT is dusted at the rate of 100–300 mgm. DDT per sq. yard in places frequented by the rats. The insecticide kills the larvae in these sites, and it is picked up in the fur of the rats and thus kills the adult fleas.

The rats are subsequently destroyed by poison baiting with "ANTU" (alpha naphtha thio-urea) or "1080" (sodium fluoracetate). The former is prepared in 1 to 2 per cent. concentration in a variety of bait formulae and sprinkled over food or water surfaces. It is the material of choice where *R. norvegicus* is the pest and where there is danger of poisoning children or animals. "1080" is a far more powerful poison, in fact one of the most toxic substances known. It should only be used by skilled operators, where the rodents are not Norway rats, and where there is no risk of poisoning other mammals.

For the protection of field workers, the underwear can be impregnated with 2 per cent. by weight of DDT, or the trouser-legs or puttees, etc., can be sprayed with modern synthetic insect repellents. *J. R. Busvine.*

CHOLERA.

PETERSON, J. S. **Epidemiological Studies in Cholera. I. Sulfadiazine Prophylaxis against Cholera. II. Duration of the Convalescent Carrier Period in Cholera. III. Secondary Attack Rate among Cholera Household Contacts, China, 1946. IV. Occupation, Age, Sex, Secondary Cases, Vaccination, Case Fatality.** *Chinese Med. J.* Shanghai Edition. 1946, Sept.–Oct., v. 64, Nos. 9/10, 271–5; 276–84, 4 figs.; 285–8; 289–95.

I. In this evaluation, not of treatment but of prophylaxis, the test and control groups were satisfactorily comparable as alternate cases and in regard to age and sex distribution. Sulphadiazine was given in daily doses to each member of the appropriate group in doses of 1 gm. for five days. The two groups consisted of 2,205 persons adequately treated and a control group of 2,563 persons. There were, however, only two cases of cholera in the treated group and eleven in the control: these results certainly suggest the possibility of sulphadiazine being a true prophylactic, but as the author declares: "Conclusions can be no more than tentative from such material".

II. The problem of the chronic carrier has not been regarded as of the same importance in cholera as in the enteric fevers. During June, July and August, an outbreak of cholera in Shanghai furnished 4,500 cases of bacteriologically proven cholera. A number of statistical tables and charts are given of the results obtained, from which we learn that of 1,949 patients whose stools showed conversion from positive to negative culture, the peak was reached on the 6th day or, nearly equally, for the 4th, 5th and 6th days. One patient continued on the positive list to the 17th day, but none thereafter. "The average number of days it took for conversion (onset of symptoms to first negative culture) was 5·4 with a standard deviation of $\pm$ 2·3".

III. Statistics of the kind set out in this communication are inevitably dependent on factors known to be exceedingly inaccurate. It is best to take the author's statement, that the comparison is not strictly valid and, indeed, is speculative, as very true. Real totals of the population at risk, weighting of sub-populations with deaths, etc., are well known as factors in causing wide divergence of ratios. We may take the deductions made as first approximations and the nearness of the approximation as very dubious. "The ratio . . . of the attack rate for general population compared to that for household contacts would appear to be . . . 1 : 36 at the most and probably even less". It is almost to be expected that attack rates among the unvaccinated will be greater than among the vaccinated in these contacts, in this case they were 5·6 times as great. What is probably true is that fatality rates show little difference between vaccinated and unvaccinated.

IV. The tables given in this fourth part of the article are careful and such as should accompany all epidemiological statistics. Very wisely the author concludes that " these observations are made without too much comment because the material itself is not too reliable and because it is felt that comment should come only after further study of the subject ". *W. F. Harvey.*

AMOEBIASIS AND INTESTINAL PROTOZOAL INFECTIONS.

DeLamater, J. N. & Hallman, F. A. **Studies on the Culture of** *Endamoeba histolytica.* *Proc. Soc. Exper. Biol. & Med.* 1947, May, v. 65, No. 1, 26–9.

With the view to determining the essential growth factors for *Entamoeba histolytica* the authors have devised a simplified medium consisting of (*a*) a buffered infusion of egg white, prepared by mixing one part of egg white with three parts N/15 phosphate buffer (*p*H 6·8) ; (*b*) sterile rice starch ; (*c*) human serum (Seitz-filtered) ; (*d*) protein-free fraction of the serum (protein removed by acidifying the serum to *p*H 5·2 and heating at 100°C.) ; (*e*) dialysate of the protein-free fraction, prepared by treating the serum with 2 volumes of 0·9 per cent. saline for 24 hours. Cultures of *E. histolytica* in various combinations of these constituents were grown in the presence of a bacterial flora comprising a diphtheroid, *Staphylococcus aureus*, *Bact. coli*, a gamma streptococcus and a Gram-variable branching bacillus which has not been identified. The authors have demonstrated that the protein-free fraction of human serum contains a heat-stable (100°C. for 4 hours) dialysable substance which is essential for the growth of *E. histolytica* in egg white buffer infusion medium containing starch. It was also found that partially deproteinized filtrate of human serum diluted in an equal volume of the buffer, with addition of starch, represents a simple medium for the cultivation of the amoeba. Rice starch appears to have an inhibitory effect on bacterial multiplication, resulting in better growth of the amoebae. *C. A. Hoare.*

Balamuth, W. **Improved Egg Yolk Infusion for Cultivation of** *Entamoeba histolytica* **and other Intestinal Protozoa.** *Amer. J. Clin. Path.* 1946, June, v. 16, No. 6, 380–84.

The author describes a standardized medium for the cultivation of *Entamoeba histolytica* and other intestinal protozoa, with the use of dehydrated egg yolk instead of fresh eggs. A comparison of dehydrated yolks from different sources has shown that the best results were obtained with material which had been processed and dried from perfectly fresh eggs. When kept in cold storage at 4°C., the growth-promoting properties of dehydrated yolks is retained at least for one year, while at room temperature they are deteriorated within a few weeks. The medium is prepared as follows : (1) 36 gm. of dehydrated egg yolk are mixed with an equal amount of distilled water and 125 cc. of 0·8 per cent. solution of NaCl are added to the mixture, which is then stirred vigorously with a rotary beater. (2) The mixture is heated in a covered double-boiler for 20 minutes after the temperature of the infusion has reached 80°C. After heating, loss by evaporation is compensated by addition of distilled water (usually about 20 cc. are required). (3) The product is then filtered by expressing the extract through a double layer of muslin, the average yield being 90–100 cc. of a yellowish fluid ; this volume is brought up to 125 cc. by addition of 0·8 per cent. solution of NaCl. (4) The extract is then autoclaved at 15 lb. pressure for 20 minutes, after which a yellowish sediment separates from the

transparent fluid. (5) The extract is cleared by cooling it below 10°C.; this may be followed by filtration with suction through a Buchner funnel, by means of a medium grade filter paper (Whatman No. 2), but the latter process is not essential. (6) To the filtrate is added an equal volume of M/15 potassium phosphate buffer adjusted to *p*H 7·5±. (7) Powdered Wilson liver concentrate (or Lilly's liver extract) 1-20 in concentration of 0·5 per cent. is added to the buffered infusion. Liver extract ensures a more rapid growth and is employed chiefly for use in clinical diagnosis but is not necessary for the maintenance of stock cultures. (8) The medium is finally autoclaved in tall test tubes and is stored in the refrigerator until required, when it is dispensed into ordinary test-tubes in amounts of from 7 to 10 cc. Before inoculation a loopful of sterile rice starch is added to each tube. The final reaction of the medium is about *p*H 7·3.

The author further discusses the effects of varying the conditions of cultivation and of modifications in the constituents of the medium. It was found that medium prepared from whole egg was not superior to yolk alone, whereas egg-white alone did not support growth. For these reasons yolk alone was selected for the standard medium. Though the concentration of yolk recommended in this paper proved to be the most efficient, growth of *E. histolytica* was also obtained in media containing half and twice the usual concentration of yolk. It is noted that the standard medium contains the requisite kinds and amounts of inorganic ions, some of which (sodium, potassium, chlorides and phosphates) are introduced in the form of salts, while calcium is derived from the yolk. As regards the reaction of the medium, *E. histolytica* grows in the range from *p*H 6 to *p*H 7·6, but experience has shown that when cultivated from stools the amoebae thrive best in competition with the accompanying bacterial flora, when the reaction is neutral or slightly alkaline.

Though primarily adapted for the cultivation of *E. histolytica*, the new medium is also suitable for other intestinal protozoa, *e.g. Entamoeba coli, Dientamoeba, Endolimax, Iodamoeba, Trichomonas* and *Chilomastix.*

C. A. Hoare.

FAUST, E. C., HEILBRUNN, Ilse, LEWIS, Ruth & MURRAY, Mary L. **Differences in Culturability, Infectivity and Pathogenicity of Human Strains of** *Endamoeba histolytica. Proc. Soc. Exper. Biol. & Med.* 1946, Nov., v. 63, No. 2, 270–76.

The authors have made a study of *E. histolytica* with the object of determining the correlation between its culturability, infectivity and pathogenicity, using strains isolated from 18 persons in New Orleans, 12 of whom suffered from intestinal disorder, 4 had other complaints, one was a symptomless carrier, while of another no history was available. Cultures were grown in a medium with solid egg-slant covered with saline containing 0·5 per cent. liver extract, to which rice starch was added. Infectivity and pathogenicity were tested by inoculation of kittens, the results being assessed on clinical evidence and at autopsy. Of the 18 strains, 6 proved difficult to grow in culture; and 4 of these were unsuitable for animal inoculation. All the other strains were grown successfully in culture and, except two of them, produced experimental infections in kittens. The results of these tests showed that culturability, infectivity and pathogenicity are not necessarily correlated with each other, but represent three separate attributes of *E. histolytica*. Thus, there may be difficulty in establishing an infection in kittens with strains growing readily in culture, or a highly pathogenic strain has a low degree of culturability. Furthermore the infectivity of a strain for kittens is not always correlated with symptoms and lesions; thus, strains with low infectivity proved to be highly

pathogenic, while very infective strains produced slight lesions. Moreover, the clinical symptoms in the experimental host did not coincide with the extent of damage revealed *post mortem*. From this and other studies of the same kind, it is evident that further investigations are required to determine the factors which are responsible for the development of the parasite *in vitro* and *in vivo*.

C. A. Hoare.

BEEMER, A. M. **Supra-Vital Staining of Amoebae and some Observations on the Laboratory Diagnosis.** *South African Med. J.* 1947, Aug. 9, v. 21, No. 15, 550–54. [11 refs.]

Prompted by the realization that the diagnosis of amoebiasis often presents great difficulty, recourse has been had to a supra-vital staining method to facilitate identification of amoebae. A method of obtaining a "satisfactory" specimen is outlined as follows:—a saline purge of magnesium sulphate is given at 7 a.m.; should an unsatisfactory result be secured a second and larger dose on the following day will succeed. Chemical irritants and oils must be avoided.

With the object of overcoming difficulties in the differentiation of amoebae and cysts the following supra-vital staining method was devised.:—

Solution A.		
Brilliant cresyl blue	0·2	gm.
Sodium citrate	1·1	gm.
Sodium chloride	0·55	gm.
Hydrarg perchlor. (sat. sol.).	0·1	cc.
Water	100	cc.
Solution B.		
Eosin	1	gm.
Normal saline	100	cc.

Equal quantities are mixed and one loopful of the mixture is added to a suspension of faeces on a slide.

Amoebae and flagellates in the living state appear as clear, translucent, pale blue-green objects against a pink background. Motility is unimpaired. Dead amoebae and flagellates take up the pink stain and the nuclei become quite distinct. By increasing the proportion of solution A, it can be shown that *E. histolytica* absorbs particles of cresyl blue.

For fixed film preparations, good results are obtained by a modification of Schaudinn's method in which solutions A and B are used in place of iron haematoxylin. The details of this modification are described.

In these preparations, amoebae and cysts usually stain pale carnation pink. Macrophage cells can be distinguished by their multicoloured granules and the large nuclei. The vacuole of *Iodamoeba bütschlii* becomes distinct. *E. histolytica* appears to ingest objects and matter other than red blood corpuscles.

A satisfactory warm stage can be improvised by pinning a cloth or duster around the draw-tube of the microscope above the coarse adjustment rack and draping the cloth over the lower part of the microscope and lamp (80 kilowatt). By these means, some specimens of *E. histolytica* remain motile after 36 hours when in wet preparations treated with vital stain, despite the presence of mercuric chloride in the stain.

P. Manson-Bahr.

ROBERTSON, K. M. **Acute Amoebiasis complicating Cholecystectomy. Report of a Case.** *Lancet.* 1947, Sept. 6, 355–6.

This case relates to a man of 37 who had been in Northern Rhodesia for many years and whose history included a severe attack of blackwater fever, complicated by appendicitis, which had occurred in Britain 13 years previously.

Since the operation for appendicitis the patient had been in excellent health: he was not aware of any previous dysentery, but occasionally suffered from short attacks of diarrhoea, usually lasting only one day.

In September 1946, he was admitted to hospital for cholecystectomy for symptomless calculi, in view of his pending return to Africa. At operation, gallstones and biliary sand were found, the fundus of the gall-bladder was buried for about an inch in liver substance and its removal required a small incision into the liver tissue; the resulting small cavity was carbolized before being closed. The liver and all the colon seen were apparently normal.

The patient progressed well until the morning of the fourth day, when he had several loose stools. His temperature rose to 100°F. and thereafter bowel action increased in urgency, and the temperature continued to rise. Several examinations of stools for amoebae and cysts were negative and blood slides were negative for malaria. The white cells were 14,000 per cmm., with a relative increase of polymorphs.

The patient became rapidly more ill, and ascites developed, with a tender abdomen. The diarrhoea resisted full doses of succinyl sulphathiazole. No signs of suppuration could be found, though the rectal wall felt indurated and painful.

The abdomen was re-explored through the original wound: no subphrenic or pelvic suppuration was found, but much bloodstained fluid escaped from the peritoneal cavity. It was found that the colon was intensely inflamed and thickened, especially in the ascending colon and hepatic flexure; the gut was accidentally torn in this region during exploration. Microscopical examination of the escaping faeces showed typical vegetative forms of *Entamoeba histolytica* in large numbers. Drainage was established and the wound closed; the patient was given transfusion of blood, followed by glucose and glucose-saline for four days.

Emetine was given in 1 grain doses every 12 hours for three doses, and subsequently daily until 12 grains had been given. Penicillin in 100,000 unit quantities was injected every 3 hours for a week. The patient remained very ill until the end of the week and then recovered steadily. As soon as fluids were allowed by mouth, he was given 8 gm. of succinyl sulphathiazole every 8 hours.

After the emetine injections, E.B.I. was given, followed by a course of carbarsone. The patient, who had developed a small faecal fistula which has since healed, has shown no further evidence of residual dysenteric infection.

The author comments that a "cold" cholecystectomy seems to have caused a flare-up of an unsuspected amoebiasis. It is suggested that the complication was influenced by trauma in the big bowel by retractor pressure, by the incision and carbolization of the cavity in the liver, and by the general lowered resistance from surgery. It is pointed out that, whatever the causal factors, they might appear in any abdominal operation. *H. J. O'D. Burke-Gaffney.*

JONES, W. R. **The Therapeutic Action of some Known Amoebicides in Rats.** *Brit. J. Pharmacol.* 1947, Sept., v. 2, No. 3, 217–20, 3 figs.

The author has recently described a test for amoebicides in which rats are inoculated intracaecally with cultures of *E. histolytica* [this *Bulletin*, 1947, v. 44, 313]. After treatment with drugs, the degree of infection is assessed in control and treated animals. With the same technique he has now compared the amoebicidal activity of emetine, stovarsol, carbarsone, chiniofon and diodoquin, drugs which have been used with success in clinical practice. Such comparative tests should be useful in screening new chemotherapeutic agents. The drugs were given in one schedule, orally in a single dose 24 hours after infection and, in the other, in multiple doses at intervals of 24, 30, 48, 54, and

72 hours thereafter, and the animals were killed six days later at which time control animals show the maximum degree of infection. In assessing the degree of infection, six grades of intensity were arbitrarily selected and each was allotted a number. The average degree of infection (ADI) in a group of animals was then calculated. The results were treated statistically. Attention is drawn to possible differences in the nature of the infection in man and rats with regard to treatment. The results with rats appeared to agree reasonably well with those obtained during the acute stage of the disease in man. " Emetine and chiniofon appeared to have the widest range of activity. Stovarsol, carbarsone, and diodoquin were effective when given in large doses."

J. D. Fulton.

WENRICH, D. H. **Culture Experiments on Intestinal Flagellates. II. Additional Observations on Flagellates from Man, Rodents and Insect Larvae.** *J. Parasitology.* 1947, Feb., v. 33, No. 1, 25–8.

This is a continuation report of the results of cultivation of intestinal flagellates by a new method [see this *Bulletin*, 1947, v. 44, 91]. Using the same media, the author obtained survival of human strains of *Trichomonas hominis* up to 114 days at 32–33°C., while *Embadomonas intestinalis* lived for 244 days at room temperature. Cultures of Trichomonads from rats were positive after 583 days, while those from hamster and from the faeces of a man, who had swallowed a culture of the hamster Trichomonad, survived for over 2 years. Cultures of a *Monocercomonoides* from a pocket gopher persisted for 484 days, while a species of the same flagellate isolated from guineapigs lived up to 633 days, and one from beetle larvae survived up to 505 days. *C. A. Hoare.*

RELAPSING FEVER AND OTHER SPIROCHAETOSES.

MERSKEY, C. **Relapsing Fever in Cullinan (Transvaal) with a Short Reference to Penicillin Therapy.** *Clin. Proc.* Cape Town. 1947, June, v. 6, No. 4, 113–24, 1 fig. [20 refs.]

A record of 41 cases of tick-borne relapsing fever treated in a Military Hospital in Cullinan, during the period July 1944, to June 1945. Only one of the patients was European, the explanation being that ticks abound in native hutments but are rarely seen in well-kept European quarters.

The results emphasize the difficulty of treating this type of relapsing fever. Arsenicals, including N.A.B. and mapharside, were found to be of very little value, and, in the case of penicillin, doses of under 1 million units had no effect in 2 out of 3 cases studied. *E. Hindle.*

ANDERSON, T. F. **Tick Control by Gammexane.** *East African Med. J.* 1947, July, v. 24, No. 7, 259–61.

Until recently it was believed that *Ornithodorus savignyi* was the sole or principal vector of relapsing fever in British Somaliland. Now, however, *O. moubata* has been found to be very common, and is usually found in houses and coffee shops. *O. savignyi*, on the other hand, is commonly found out of doors in places where camels and other stock have halted.

The author studied the use of Gammexane (666) in the control of *O. moubata* in 16 coffee shops chosen at random. Four other such shops were untreated and served as controls. Searches for ticks were made in each case over an area

covering 10 feet of the floor near the walls and surrounding the bases of roof-poles. Only those ticks large enough for certain immediate identification were included in the catches. After identification the ticks were replaced in their former situations.

The premises were examined at two fortnightly intervals after the first search and again one month later. After each search, 3 per cent. Gammexane in diesoline was applied to the whole floor area with a pressure sprayer (" Four Oaks Kent "), so that 600 mgm. of Gammexane were applied per square foot. Control shops received no treatment.

Tables in the text indicate that 9 of the 16 shops contained ticks at the first search, the average number being 10 per house ; at the second search this average was reduced to 0·75 ; at the third no ticks were found ; and at the last search only one tick was present.

The corresponding figures in the text for the four control shops are given as 18, 36, 12 and 6 ticks [these figures do not correspond exactly with those given in the table].

It is, however, clear that, in these experiments, the number of ticks was greatly reduced after the first application of Gammexane and that the second application almost completely eliminated them, while the reduction in the untreated shops, where this could be examined, was comparatively slight.

H. J. O'D. Burke-Gaffney.

Albacete, R., Curbelo, A. & Florin, Maria C. Un caso de leptospirosis ictero-hemorragica simulando una ictericia catarral. [**A Case of** *Leptospira ictero-haemorrhagiae* **Infection simulating Catarrhal Jaundice.**] *Vida Nueva.* 1947, Mar., v. 59, No. 3, 113–25, 4 figs. [21 refs.]

LEPROSY.

Muñoz Rivas, G. Pulgas, suelos y lepra. [**Fleas, Soil and Leprosy.**] Primer Congreso Inter-Americano de Medicina, 7 a 15 de septiembre de 1946, Rio de Janeiro. 51 pp., 11 figs. [69 refs.]

This interesting article is, in essence, an amplification of work published by the author in 1942 [this *Bulletin*, 1943, v. 40, 151]. He considers in turn the following points : (i) *Mycobacterium leprae and the larvae of fleas.* Among 341 captured in 121 leprosy dwellings he found acid-fast bacilli indistinguishable from Hansen's bacillus, in the stomachs of 142, or 41·6 per cent., but none in 506 larvae captured in healthy dwellings, at least in dwellings not inhabited by lepers. (ii) *Mycobacterium leprae and adult fleas.* Among 1,627 fleas (*P. irritans*) which had fed on leprosy patients, 187 (11·4 per cent.) were positive, whereas none was found positive among 575 fleas captured in sites presumably free from infection of leprosy. Further, of 338 adult *P. irritans* bred from experimentally infected larvae, 8 (2·3 per cent.) had the organisms in their stomachs, whereas none was found among 177 fleas bred from uninfected larvae. (iii) *Does the bacillus undergo evolution in the digestive canal of P. irritans?* No definite evidence of this has been obtained. (iv) *Inoculation trials.* Inoculation of *young Macaca* and *Cebus* monkeys with triturated fleas which had fed on leprous patients, or with insect dejecta or triturated fleas bred from infected larvae, caused gland enlargement and temporary presence of acid-fast bacilli, and in two of the monkeys an increase in rate of corpuscle sedimentation. (v) *Incidence of leprosy and prevalence of fleas.* Among 3,399 cases reported in

17 Departments of Colombia, in Santander with 1,052 cases, Boyaca with 954, Cundinamarca with 497, fleas are recorded as "very numerous"; in North Santander with 236, as "numerous"; in others, with 108 or less, they are "scarce". (vi) *Do other insects play a part in the transmission of leprosy?* Ticks, lice, mites, bugs, Simuliidae, other diptera, and flies are discussed briefly, with references to the literature, but no first-hand evidence is adduced. (vii) *On acid-fast bacilli and attempts to cultivate them from the soil and from flea larvae in dwellings inhabited by lepers.* Remarks are made on certain organisms thus cultivated.

This article is of interest as a further study tending to incriminate the human flea, *P. irritans*, as *a*, if not *the*, vector of *Mycobacterium leprae*. Worthy of note also is the statement of Dr. Juan de D. CARRASQUILLA who, in 1905, wrote: "Leprosy is not contagious in the same way as diseases such as small-pox; it is infective and the agent is the flea." (La lepra no es contagiosa a la manera de otras enfermedades . . . como la viruela; es infectiva y el agente de infección es la pulga.) *H. Harold Scott.*

ROSSELL, C. S. C. Cultura cromogênica de um bacilo ácido-álcool resistente, isolado de mosquito (Culicíno) capturado sôbre leproso, em plena natureza, com rétrocultura obtida de rato branco. [**Culture of an Acid-Fast Bacillus from a Mosquito caught on a Leprosy Patient, with Subculture from a White Rat.**] *Mem. Inst. Oswaldo Cruz.* 1946, Sept., v. 44, No. 3, 413–24, 7 figs.

DE SOUZA-ARAUJO, H. C. **Clamp Method to obtain Cutaneous Lymph in the Diagnosis of Leprosy.** *Leprosy Review.* 1947, Apr.–July, v. 18, Nos. 2/3, 44–5.

The author describes a modification of the Lleras method of obtaining skin lymph, which he has used successfully in Brazil.

The area of skin is gripped with Pean's haemostatic clamp, which is tightened according to the thickness of the skin. The area becomes quite ischaemic within one minute. It is then punctured deeply at four points with a large needle and four drops of clear lymph exude, which are smeared on a clean slide with a vaccination pen. The slide should be covered and left to dry for a few hours and then stained by the Ziehl-Neelsen method. This modification, by obtaining four samples of lymph, increases the chance of finding *Myco. leprae*.

Where there is dense subcutaneous tissue, *e.g.* in the back or buttocks, two clamps are used.

In diffuse lepromatous lesions, enormous numbers of acid-fast organisms are found and they stand out clearly, in the absence of tissue elements.

The author was able to find bacilli in all cases of tuberculoid leprosy, even in bundles or globi, by this method where ordinary biopsy specimens failed to show them. He has found the Lleras technique excellent for examination of cases in general control of treatment and parole.

H. J. O'D. Burke-Gaffney.

DE OLIVEIRA CASTRO, G. M. **Staining Nodules of the Leprosy Bacillus.** *Leprosy Review.* 1947, Apr.–July, v. 18, Nos. 2/3, 45–9, 2 figs.

The author uses the term "bacilli nodules" or "nodules" for the rounded spore-like thickenings seen in lepra bacilli: an unfortunate term. His method is a modification of that of Cooper, in which the addition of sodium chloride to a solution of carbol-fuchsin produces a precipitate at room temperature, which redissolves on heating. The present writer prefers the addition of 10 per cent. solution of potassium phosphate, KH_2PO_4, 3 cc. of which is added to 100 cc. of carbol-fuchsin, or 5 drops to 10 cc. just before staining. Flood the slide with

this stain, steam for five minutes and allow to cool until a cloudy precipitate of the stain appears, and pour off the stain. Without washing, decolorize in 3 cc. HCl in 97 cc. of 96 per cent. ethyl alcohol until the dye ceases to flow off. Counter-stain for half to one minute with a dilute solution of 0·1 gm. methylene blue in 1,000 cc. water. Wash and dry. The acid-fast bacilli are stained red and the "nodules" within them stain very dark red or brown, as spherical structures of a larger size than the bacilli; they may be at the ends of the bacilli and usually number 1 to 3 and rarely exceed 4 or 5. They are present in practically all the bacilli. Similar results are obtained by staining tubercle bacilli in sputum. *L. Rogers.*

MUIR, E. **Classification of Leprosy Cases.** *Leprosy Review.* 1947, Apr.–July, v. 18, Nos. 2/3, 73–82, 1 diagram.

The importance of a general adoption of a sound classification of leprosy cases is stressed and the South American division into the two characteristic lepromatous and tuberculoid types—together with a third uncharacteristic chronic inflammatory one, which may later develop into one of the typical ones—is discussed and illustrated by a diagram. This represents a doorway below into the intermediate uncharacteristic form, which leads off laterally into lepromatous on one side and tuberculoid on the other. The well-recognized microscopical characters of the typical forms and the positive lepromin test in tuberculoid and negative ones in lepromatous cases is stressed. The third intermediate type is subdivided (*a*) initial, in the course of passing into one of the typical forms; (*b*) intermediate, passing from one of the characteristic types into the other; or (*c*) vestigial, passing out of one of the characteristic types on the way towards recovery. The chief indication of an uncharacteristic case is one of exclusion, its failure to belong to either of the other two types. The lesions are usually flat and thin and without the thickening of the skin of lepromatous cases, and they require a good light for recognition. Bacilli are few or nil in them and the lepromin test negative or moderately positive, while they rarely show a reactionary phase. The author holds that tuberculoid cases may be transformed into lepromatous ones, but only infrequently. The reverse change is of more doubtful occurrence. The paper concludes with a list of points to be noted in making a clinical examination, together with forms for case taking, which should be read in full by those interested. *L. Rogers.*

COCHRANE, R. G. **Child Leprosy.** *Leprosy Review.* 1947, Apr.–July, v. 18, Nos. 2/3, 49–53.

This article was written for a special number of *Leprosy Review*, in connexion with the retirement of Dr. E. MUIR. After paying tribute to the long and valuable work of Muir, the author records once more his well-known views on child leprosy and states that he is in agreement with early emphasis laid on the importance of the subject by Rogers and by Muir. Only a certain amount of child leprosy is serious from the preventive aspect in South India, for a significant proportion of cases are benign and non-progressive. Practical consideration need only be given to (1) Simple neural leprosy, and (2) Pre-lepromatous lesions, the latter comprising perhaps only five to ten per cent. of all child cases, which develop into lepromatous leprosy late in life and mostly arise in families where there is the closest contact from early life with open cases. These cases require closer attention in order effectively to control them. He thinks that the corium of the skin is the site of active development of lepra bacilli and that those in the reticulo-endothelial system are mainly saprophytes living in a state

of commensalism. The benign cases in children do not require prolonged treatment, but every effort should be made to prevent the occurrence of deformities. *L. Rogers.*

PARDO-CASTELLO, V., TIANT, F. R. & PIÑEYRO, R. **Nerve-Lesions of Leprosy.** *Arch. Dermat. & Syph.* 1947, June, v. 55, No. 6, 783–9, 5 figs.

The pathological and bacteriological aspects of leprosy as it affects the peripheral nerves is dealt with. In a previous paper, the authors [this *Bulletin*, 1943, v. 40, 785] insisted on the presence of nerve lesions in almost every case of leprosy, and on the importance of distinguishing the lepromatous from the tuberculoid types. They also pointed out that the structure of the lesions of the nerves coincides with that of the lesions of the skin.

These points are further emphasized in the present paper. In lepromatous cases, the extensive skin lesions are accompanied by clinical involvement of large trunks such as the ulnar, median and saphenous nerves more especially; but the most remarkable clinical neuritis is found in tuberculoid leprosy, both in conjunction with skin lesions and at times almost exclusively affecting enlarged terminal nerves and nerve trunks, with cutaneous anaesthesia and muscular atrophy. Sections of ulnar nerves in lepromatous cases show cellular infiltration throughout, including the perineural tissues, together with atrophy of the nerve fibres and the presence of enormous numbers of lepra bacilli. On the other hand, in tuberculoid cases both peripheral and trunk nerves show thick infiltrations of lymphocytes, epithelioid and giant cells, but few or no lepra bacilli as in tuberculoid lesions of the skin. Caseation or abscess formation may result, with complete destruction of the neural tissues. The authors are therefore of the opinion that the so-called neural leprosy belongs to the tuberculoid type, with which it also corresponds bacteriologically and immunologically in giving positive lepromin reactions.

In the discussion Dr. Braulio Sáenz expressed disagreement with this view. *L. Rogers.*

MOM, A. M. Benadryl en la reacción leprosa lepromatosa y en la sensibilización sulfonica. [**Benadryl in the Treatment of the Leprous Reaction.**] *Rev. Argentina Dermatosifilologia.* 1947, June, v. 31, No. 2, 188–91.

Allergic reactions, of which the leprous reaction is one, are due, it is said, to a substance closely allied to histamine, "probably identical with it." It is argued, therefore, that the leprous reaction may be modified, or even nullified, by synthetic anti-histamine substances. Benadryl, the hydro-chloride of benzhydryl diethylaminoethylic ether, has, therefore, been tried in six cases of the lepromatous form with the leprous reaction; two were women, 22 and 24 years of age, and four were men between 24 and 42 years, and all were moderate cases of 1½ to 8 years' evolution. Each of these received 150–200 mgm. of benadryl during the day in capsules of 50 mgm., for 8–10 days. In four of the patients, marked change for the better was seen in 5–8 hours; the pain was less, temperature fell, erythema was reduced and soon faded altogether. The response was so rapid that in two of the patients the drug was stopped after 6 days, and during the ensuing 30–70 days there was no relapse. In two others, there was no such beneficial action and to one there was given 100 mgm. daily of thiamine chloride and tablets of acetosalicylic acid. Apart from the quick relief of symptoms, there is the advantage that it permits early resumption of specific treatment, such as promin or chaulmoogra, without risk of fresh leprous reaction. Benadryl also counteracts sensitivity to sulphonamide drugs, relieving the pruritus, urticaria and other rashes. It is to be noted that all the patients had the lepromatous type of the disease. *H. Harold Scott.*

COCHRANE, R. G. **Sulphone Treatment of Leprosy.** [Correspondence.] *Brit. Med. J.* 1947, July 19, 110–11.

This letter is written to protest against " a tendency for treatment to receive precedence over prevention " and to sound a warning note against the belief, apparently held in some quarters, that " the leprosy problem could be solved by curative measures alone." [The author does not indicate who holds such a view, nor can the reviewer think of one such in his extensive acquaintance with the literature.] The author goes on to state the well-known facts that promin is toxic and diasone may produce reactions, so both should only be used at present under carefully controlled conditions. He further states that sulphetrone is the least toxic of this class of drugs, but that it is not likely to be on the market for some time. *L. Rogers.*

PEYRI, A. La lepra en Monterrey. Datos y comentarios. La diaminodifenilsulfona en cinco casos. [**Leprosy in Monterrey. Treatment by Diaminodiphenyl Sulphone (Promin).**] *Sugestiones.* Mexico. 1947, June, v. 12, No. 142, 22–41. 2 maps.

Monterrey is a town of Nuevo León, a province of Mexico. The author, who is a professor of the Nuevo León University, tells of his experience of leprosy after 5 years' residence. Nuevo Leon has a population of 541,147 and the total number of leprosy cases seen in the five years, 1939–44, is given as 44, or 0·8 per mille. Figures are given of previous censuses of the country. In 1927, among 15,151,695 inhabitants there were 1,450 registered cases of leprosy, 22 in Nuevo León ; in the quinquennium 1930–34 there were 249 registered, 52 in Nuevo León ; between 1935 and 1938 a total of 2,696, of which 14 were in Nuevo León ; in the five-year period 1939–44, there were 1,988 registered, 44 in Nuevo León, as stated above.

The author has no reliable data on the prevalence in Monterrey itself. There has been no register kept, nor clinical histories at the hospital, although on several occasions leprosy patients have been admitted, and he says " I have gathered from a nurse that in 8 years there have been some 10 or 12 patients." He, therefore, limits his remarks to 30 cases which he has had under his personal care and he estimates [? guesses] that the total in Monterrey during the five years is 50. Of his 30, 17 were males, 13 females ; two were under 10 years of age and the numbers in succeeding decades were 4, 7, 7, 6 and 4. Five were married, but in no instance were husband and wife both infected, and none of the 30 was aware of having lived with a leper or resided in a focus of high endemicity ; most lived in the country and were not overcrowded. Of his 30, sixteen were of the lepromatous form, four were tuberculoid and ten " not characteristic". [Later he states " 17 were lepromatous, 13 neural ".] Their environment, sanitarily, was deplorable ; there is no control at all, they do what they like where they like, they wander about unchecked, cross the frontiers and recross them unquestioned.

The first symptom noted was hypochromic blotches (5), nodules (5), patches of anaesthesia (5), epistaxis (4), and other signs in smaller numbers. To five of the patients, the author gave diaminodiphenyl sulphone (DDS) [Promin] intravenously, in doses of 2 gm. daily for six days in the week, with an interval of 15 days every two months. In all of them there was improvement after a total dosage of 200–300 gm. ; in two, bacteria were no longer found, but the drug did not prevent the appearance of a leprous reaction.

H. Harold Scott.

HELMINTHIASIS.

TALICE, R. V. Pseudoparasitismo en gastroenterología. [**Pseudoparasitism in Gastro-enterology.**] *An. Facul de Med. Montevideo.* 1947, v. 32, Nos. 1–2–3–4, 1–24, 36 figs.

The uninformed laity are repeatedly bringing to medical men specimens of faeces or substances taken therefrom which they think are parasites passed in their stools, and fear that they are harbouring others. Some of these are so like parasites that at first view they " may deceive even the very elect." The author puts on record several of these pseudoparasites and, of more value than mere description, depicts them in clearly reproduced photographs. We cannot take note of all of them here : the original should be consulted and will well repay perusal. The author divides them into two main groups : macroscopic and microscopic and subdivides each again into those of animal nature, those of vegetal nature and those of human intestinal origin. Among the first subdivision of the macroscopic group are earthworms, which are seen in faeces deposited on the ground or which have crawled into linen set out on the ground to dry, larvae of flies, *Musca*, *Anthomyia*, *Sarcophaga*, Syrphidae, etc., and free-living helminths, as *Gordia*. Of vegetal nature, cellulose residues of citrus fruits, banana fibres, badly cooked and undigested vermicelli, tangled threads of cotton which have been inadvertently swallowed. The third subdivision would include rolled masses of mucus in mucous colitis, or membranous fragments in mucomembranous colitis.

Of microscopic pseudoparasites, he mentions acarines, *Tyroglyphus* flour mites, and cysts in the liver of parasitized sardines ; of the second subgroup, epidermal residues of fruits, spores of moulds parasitic on vegetables and fruits, yeasts, etc. Of human origin, leucocytes and mucosal cells mistaken for amoebae [a mistake with which all teachers of tropical pathology and parasitology are constantly confronted by students]. *H. Harold Scott.*

JACQUES J. J. C. Effets d'une cure de vingt grammes de sulfamidés " per os " sur les verminoses intestinales de l'indigène. [**Treatment of Intestinal Worms in Africans with Twenty Grammes of Sulphonamides.**] *Ann. Soc. Belge de Méd. Trop.* 1947, Mar. 31, v. 27, No. 1, 83–4.

The treatment was without effect.

RHODESIA, SOUTHERN. DEPARTMENT OF PUBLIC HEALTH. Salisbury. Pamphlet No. 2 (Amended 1947). 9 pp., 3 pls. **The Story of Schistosomiasis (" Bilharzia ") and how to combat it.**

This pamphlet is evidently written for laymen, and presents in very lucid form the important facts concerning parasites, snails, symptoms, and measures of prevention, in relation to infection with *S. haematobium* and *S. mansoni*. In Southern Rhodesia both infections are common ; the snail hosts are *Physopsis africana* and *Planorbis pfeifferi* respectively. Prevention is dealt with under several heads :—Treatment of infected persons, Prevention of stream contamination, Killing of snails, Killing of cercariae, and Avoidance of contact with infested water.

It is a good pamphlet, which will appeal to intelligent people. Medical men in other countries in which these diseases occur may be interested to see how the subject is presented. *Charles Wilcocks.*

IGNACIO BALDO, J., GIL YEPEZ, C., MAYER, M. & PIFANO C., F. Investigaciones concernientes a los aspectos pulmonar y cardiovascular de la Schistosomiasis Mansoni en una area endémica del pais. [**Pulmonary and Cardio-vascular Lesions in Infestations with** *Schistosoma mansoni* **in an Endemic Area.**] *XII Conferencia Sanitaria Panamericana. Cuadernos Amarillos. Publicaciones de la Comisión Organizadora. No. 9.* Caracas. 1946. 68 pp., 9 figs. [21 refs.]

That pulmonary symptoms, due to the passage of metacercariae or to embolism by ova, occur in infestation with *S. mansoni* is a fact well known, and radiology demonstrates shadows of an exudative type, fleeting infiltrations, miliary broncho-pneumonia, especially in the early days of infestation and disappearing in a short time. Later, there may be larger, isolated granulations due to the presence of adult worms, or the ova, coalescing to form nodules, "Bilharziomata" or pseudo-tuberculomata, with giant cells, epithelioid cells and fibrotic changes; at other times there are irritative signs with fibrinous or sero-fibrinous exudation; others, again, show oedema, atelectasis and signs of chronic pneumonia of an interstitial type, but no ova are seen and the changes are very possibly due to toxins of the parasite.

As regards cardio-vascular changes, there may be a proliferative arteritis, like endarteritis obliterans, leading to occlusion and perhaps bilharzial nodule formation, or toxic and allergic in origin.

The author decided to look into these matters more thoroughly and accordingly undertook to examine the lungs of children and persons under 20 years of age in an endemic area of schistosomiasis in Venezuela. The age was limited with a view to excluding conditions common in adults over that age. For cardiac changes children only were selected in order to exclude rheumatism, syphilis, etc., common in older persons. It is stated that tuberculous infection rate is high in the country; in the towns, 50 per cent. of those under 15 years react positively to tuberculin.

The district chosen was San Casimiro, through which run the rivers Gueripa, Toronquey and Suata and where cercarial dermatitis, "pica-pica", is common. The intradermal schistosome-antigen reaction was tried in 340 subjects; the results are presented in a table [the numbers are small, and the results as stated seem somewhat biassed, the "doubtful" being counted as positive]. Of these 165 were positive, 21 doubtful, and recorded as 186, or 54·7 per cent., positive; it was highest, 18 positive and 2 doubtful, among 24 rural schoolchildren. Only 156 faecal examinations were carried out (by Stoll's method); of 42 town-dwellers [? adults] 21 were positive, of 81 children attending town schools 28 were positive, and 15 of 33 attending rural schools. Other infestations, as by *Ascaris*, *Trichuris* and *Necator* were common; *Taenia* less so.

Of 179 chests radiologically examined [another 9 screened are not discussed], 97 were normal, 82 showed some change: lungs "atypical" in 48, small nodules in 11, residual pleurisy in 4; but three of the last and several of the former gave a positive tuberculin reaction.

Cardiac enlargement was seen by X-rays in 28; in 11 a general enlargement, in 6 enlargement of the left side, in 9 of the right, and in 2 a "globular heart". Clinically, it is said, the findings were of little importance, but dyspnoea and palpitation on slight exertion were common, with accentuation of the pulmonary second sound. The lesion was considered to be of the nature of a myocarditis. [Reproductions of the X-ray photographs are given, but are printed too darkly to be very informative.] *H. Harold Scott.*

RITCHKEN, J. & KANTOR, F. **Herpes Zoster. A Toxic Manifestation resulting from the Therapeutic Administration of Antimony and Potassium Tartrate in the Treatment of Schistosomiasis.** *Clin. Proc.* Cape Town. 1947, June, v. 6, No. 4, 125–9.

Since July 1945, the authors have treated schistosomiasis by the intensive method of ALVES & BLAIR [this *Bulletin*, 1945, v. 42, 815; 1946, v. 43, 344] in preference to the previously used administration of smaller doses over a longer period.

In 5 of 105 patients so treated, a typical attack of herpes zoster, indistinguishable from "idiopathic" herpes, developed between the 5th and the 10th days after completion of treatment: no children were affected. All patients recovered without sequelae, on symptomatic treatment. The delayed onset of herpes was not related to the severity of the initial toxic symptoms, *e.g.* while some coughing and vomiting followed at least one (usually the first) injection in most cases, this tendency was no greater in those who subsequently developed herpes. The doses given varied from 10 to 14 grains. In three of the cases, a mild, exfoliating palmar erythema occurred: in one case, the eruption was generalized at first. Such eruptions were also seen in many cases where herpes did not occur.

The authors discuss the incidence and aetiological theories of herpes zoster, and draw freely on existing literature. They note its association with arsenic, bismuth and other chemicals, but are unable to find any record of the condition following the administration of antimony. The current consensus favours the belief that all herpes zoster is due to a virus and that in "symptomatic" cases this is activated by the particular factor concerned as the resistance of the nervous tissue is lowered.

In view of the similar chemistry of antimony to that of arsenicals, which are well known to be followed at times by herpes zoster, the occurrence of such a reaction as a result of the administration of antimony is not surprising. It is suggested that the virus of herpes zoster is very commonly harboured by man, and the infection may result in carriers from any process tending to lower resistance in the spinal ganglia.

The authors reviewed some 200 records of treatments of schistosomiasis by the well-known routine administration of ½–2 grains of antimony and potassium tartrate over long periods. Only one patient was found in whom herpes zoster had developed during treatment. It is therefore suggested that the rapid treatment does render the patient more liable to the development of herpes zoster, although the total dosage of antimony is about half of that used in the older method.

[See also "Arsenical Chickenpox"—*Bulletin of Hygiene*, 1946, v. 21, 794.]

H. J. O'D. Burke-Gaffney.

LUTTERMOSER, G. W. La campaña antibilharziana en Venezuela. [**The Campaign against** *Schistosoma mansoni* **Infestation in Venezuela.**] *XII Conferencia Sanitaria Panamericana. Cuadernos Amarillos. Publicaciones de la Comisión Organizadora. No. 12.* Caracas. 1946, 74 pp., 20 figs. & 3 folding pls. (2 maps). [Bibliography.] English summary.

An interesting account of what has been done to overcome schistosomiasis in Venezuela during the past 30 years, since the time when Iturbe and Gonzalez discovered the snail vector. As a result of this, recommendations were made to drain infected land bordering on the streams, to dry up water collections temporarily, to apply lethal chemicals and to establish adequate latrines. From 1941 measures have been more energetically pursued. The Ministry of

Health then took up the question in earnest; an anti-bilharzia dispensary was established, an anti-snail campaign inaugurated and later intensified, first in Caracas, then in Guarenas.

The present publication details the means employed in carrying these measures into effect. First comes the laboratory and associated clinic for diagnostic ends; faecal examination by the Stoll technique and, if that proves negative, by a method of sedimentation and examination of the deposit. Other methods used were the intradermo-reaction of Fairley and the fixation of complement and, as a last resort, biopsy of tissue from the rectal ampulla. The clinical syndromes were also studied, groups being divided into hepato-intestinal, hepato-splenic, intestinal, and asymptomatic. Lastly, treatment by tartar emetic in 1–2 per cent. strength injected intravenously in doses of 0·02 gm. upwards till 1·2–1·6 gm. have been given. The patient is then re-examined and, if he is still uncured, the course is repeated. Another preparation used was Fouadin or Reprodal, a trivalent compound containing 6–13 per cent. of antimony.

The endemic zone of schistosomiasis in Venezuela comprises much of the States of Aragua, Carabobo, Miranda and the Federal District—the most thickly populated area (clearly shown in a line map). The morbidity varies enormously in different parts of this area. SCOTT [this *Bulletin*, 1942, v. 39, 865] found, in one district, the faeces of 100 per cent. of women and men and 91 per cent. of the children positive; in others only a few [the percentages are given in a table, but not the numbers on which these figures are based]. The mortality figures do not seem to be reliable; many deaths ascribed by some to this infestation are notified by others as due to complications, the primary condition not appearing in the record. Thus JAFFE stated that 37 per cent. of patients with bilharziasis died as a result of the infestation, while, in 1945, in El Valle only one death per mille was officially notified as due to this disease and it is thought that many more were included under "dysentery".

The Minister of Health, apart from the diagnostic measures mentioned above, has already established dispensaries in Caracas, El Valle and Guarenas. These are not only centres for treatment, but also for propaganda and sanitary education of the people, showing them how the infection is contracted and thus how they can avoid it. Example as well as precept is employed and proper latrines are set up; one particular type of prefabricated latrine is depicted clearly [almost the only clear illustration of the 20]. Chemical treatment of streams and small water collections by sulphate of copper and by slaked lime is detailed, and engineering projects such as proper irrigation, establishment of sewerage systems and protected aqueducts are referred to.

H. Harold Scott.

CUTLER, J. G. **Schistosomiasis of the Central Nervous System.** *J. Nervous & Mental Disease.* 1946, Oct., v. 104, No. 4, 425–31. [15 refs.]

Before the war only 9 cases of schistosomiasis of the central nervous system were described in the world literature. To this number THOMAS and GAGE [this *Bulletin*, 1945, v. 42, 1018], reporting on schistosomiasis acquired during the recent Philippines campaign, added three cases. Large numbers of troops were infected with *Schistosoma japonicum* shortly after the landing on Leyte in October 1944; the danger was foreseen, but military exigencies made it impossible to take effective precautions. Another probable case of central nervous system involvement is reported.

A 23-year-old soldier was exposed to infection on Leyte, between February and July 1945, on several occasions without any immediate ill-effects. On August 5th, he noted swelling of the right eyelids and upper lip, and angioneurotic

oedema was diagnosed. This was followed by progressive weakness of the upper and lower extremities. By August 9th he was unable even to sit up in bed: he appeared confused but answered questions with a slightly slurred speech. His head jerked spasmodically from side to side; there was nystagmus on left deviation. There was a plastic-like rigidity of the upper limbs and a flaccid paralysis of the lower with a Babinski on the left. All reflexes were hyper-active except the abdominal, which were absent.

The blood showed a high eosinophilia—43 per cent. of 22,550 leucocytes; ova of *Schistosoma japonicum* were found in the stools; and the lumbar puncture, the gold curve and the Wassermann were normal.

"It seemed plausible, however, that an invasion by parasites or ova of the brain stem in its more superficial zones to include the basilar and cerebellar tracts, could account for all the neurological findings."

Intramuscular fouadin was started immediately and 75 ml. were given in 29 days. Improvement started immediately: after three weeks, the patient was able to walk about the wards, with only a slight ataxia. When the patient left hospital 2 months later, ova had not been found in the stools for 6 weeks, but he still had eosinophilia, 38 per cent. of 11,000 leucocytes.

Chest X-rays were persistently negative, but electrocardiograms, three weeks after admission and subsequently, showed an inverted T2.

The author believes that in this case the first symptoms appeared about 4 to 5 weeks after exposure. He considers that there is evidence that these precede the deposition of ova and that, therefore, in such cases immediate specific therapy is of vital importance. *L. E. Napier.*

BERTRAND. Distomatose pulmonaire ou hémoptysie pulmonaire à paragonimus. [**Pulmonary Distomatosis or Haemoptysis from** *Paragonimus.*] *Ann. Soc. Belge de Méd. Trop.* 1947, Mar. 31, v. 27, No. 1, 1–3.

Report of a case in the Belgian Congo.

FEDERICO HEINERT, J. Paragonimiasis pulmonar o distomatosis pulmonar en el Ecuador. [**Pulmonary Paragonomiasis in Ecuador.**] *Kuba.* Habana. 1947, May, v. 3, No. 5, 101–6. English summary.

Between 1921 and 1946, the first 26 cases of paragonimiasis in Ecuador were studied. The present report is an account of this condition, with detailed clinical and pathological records of some of the cases. All except one came from provinces in the Ecuadorian littoral. Eighty per cent. of the patients were cured by emetine; the remainder, who failed to respond to emetine and tartar emetic, required "endobronchial treatment".

The author considers that infection was derived from raw or improperly cooked crabs; they also suggest, without providing supporting evidence, that prawns may have played a part. *H. J. O'D. Burke-Gaffney.*

FAIN, A. Un cas de sparganose chez l'homme, deux cas de sparganose chez le serval et un cas de diphyllobothriose (*D. parvum*) chez le chacal au Congo belge. [**A Case of Sparganosis in Man, Two Cases of Sparganosis in the Serval and One Case of Sparganosis in the Jackal in the Belgian Congo.**] *Ann. Soc. Belge de Méd. Trop.* 1947, Mar. 31, v. 27, No. 1, 65–9, 1 pl.

A human case of sparganosis was seen in the eastern region of the Belgian Congo. This, as far as is known, is the first case of its kind reported from that region.

Specimens of *Diphyllobothrium* were also found in a jackal and a serval, these being respectively *D. parvum* and sparganum. [The parasites are illustrated in a photograph.] *H. J. O.D' Burke-Gaffney.*

VON BONSDORFF, B. **"Castle's Test" in Pernicious Tapeworm Anemia. *Diphyllobothrium latum* and Pernicious Anemia VII.** *Acta Med. Scandinavica.* 1947, v. 128, Suppl. 196, 456–77, 14 figs. [10 refs.]

The author's earlier work (*Acta Med. Scandinavica*, 1939, v. 100, 436, 459; 1940, v. 105, 540) [see also this *Bulletin*, 1944, v. 41, 593, 594, 678] indicates that the gastric juice of persons suffering from tapeworm pernicious anaemia contains intrinsic factor, so that the anaemia is not due to lack of this substance. Nor is there any evidence that it is due to lack of extrinsic factor. The author suggests the idea that pernicious tapeworm anaemia can be due to interference by the tapeworm with the interaction between the intrinsic and extrinsic factors. The work of HERNBERG [this *Bulletin*, 1937, v. 34, 400: see also *Acta Med. Scandinavica*, Suppl. 1941, v. 123, 255] shows that mixtures of meat and gastric juice of patients with tapeworm pernicious anaemia can induce a typical remission when they are given to patients with cryptogenetic addisonian pernicious anaemia. The question whether mixtures of gastric juice and sources of extrinsic factor can cause remissions in patients with tapeworm pernicious anaemia has not yet been studied.

The author carried out Castle's test on 4 cases of cryptogenetic pernicious anaemia and 10 cases of tapeworm pernicious anaemia. These patients were given mixtures of normal gastric juice taken from normal healthy young subjects or from suitable patients after stimulation with 12 to 16 units of insulin given intravenously; and they received extrinsic factor in the form of minced beef or yeast extract. The gastric juice was kept half an hour at *p*H 10 and then neutralized to conform with the author's studies [*loc. cit.*] of its proteolytic activity at neutral reaction. When it was not used at once, it was frozen and kept in a refrigerator. Each day, 150 to 175 ml. of gastric juice were used for making the mixtures. The source of extrinsic factor for most patients was 150 gm. of lightly roasted minced beef, but some patients received a "watery solution of 60 g. yeast extract". The meat was mixed with the gastric juice, the mixture was stirred and, after adjustment of the *p*H to 7–7·4 if necessary, the mixture was given to the patients either at once or after incubation for 6 hours at 37°C. Each patient received the mixtures for 8 days. The basal diet was the usual hospital one, but was rather poor in proteins. Clinical data of the patients are given. The results of the experiments are illustrated by graphs.

Experiment 1 shows a "very powerful" anti-anaemic effect of the non-incubated mixture of gastric juice and beef on a case of cryptogenetic pernicious anaemia, which was not surpassed by the effect of the same mixture after incubation. The other experiments show the effects of feeding, in parallel, patients suffering from cryptogenetic and tapeworm pernicious anaemia with the same preparations of gastric juice and beef, with or without incubation; and of feeding patients suffering from tapeworm pernicious anaemia with mixtures of gastric juice and either beef or yeast extract, but without the parallel cases of cryptogenetic pernicious anaemia.

The author claims that his results provide "clear and indisputable" evidence that in cases of cryptogenetic pernicious anaemia there was a "good remission" after administration of a non-incubated mixture of gastric juice and extrinsic factor; incubation of the mixtures did not strengthen this antianaemic effect. In 7 cases of tapeworm pernicious anaemia the remission was slight or absent,

whether the mixtures were incubated or not; but, after removal of the tapeworm, good remission was obtained. Two other cases gave less clear-cut results and these are discussed.

The author concludes that the administration of extrinsic factor (meat or yeast extract) and intrinsic factor (normal gastric juice) produces no significant blood remission in cases of tapeworm pernicious anaemia, but does produce this in cases of cryptogenetic pernicious anaemia. This result is the same whether the mixtures are incubated for 6 hours at 37°C. before administration or not. He suggests the possibility that the tapeworm may destroy the interaction between the intrinsic and extrinsic factors and that this may be the explanation of tapeworm pernicious anaemia. If this is true and if FORMIJNE [this *Bulletin*, 1941, v. 38, 668] is correct in his conclusion that the site of the interaction between the intrinsic and extrinsic factors is the intestinal wall, the tapeworm interferes with this interaction in the intestinal wall.

[In the second line of the author's summary the word "extrinsic" should be "intrinsic".] *G. Lapage.*

BRACHETTO-BRIAN, D. & SCHENA, A. T. Granulomatosis equinococóccica de la pleura. (Seudo-tuberculosis equinococóccica de Dévé). [**Echinococcal Granuloma of the Pleura.**] *Rev. Asoc. Méd. Argentina.* 1947, Aug. 15, v. 61, No. 611, 577–81, 5 figs. [Summary in English by the author.]

"The authors describe the first clinico-pathological observation in human pathology of 'Echinococcus granuloma of the pleura' (Pseudo-tuberculosis of Dévé) in a patient with right hydatid-pleura, probably due to a hydatid cyst of the lung.

"The authors state their reasons for modifying the name Hydatid pseudo-tuberculosis of the serosa (Dévé) and propose to name this entity 'Echinococcus granuloma of the serosa'.

"So far 'Echinococcus granuloma of the serosa' had only been reported in pericardium and peritoneum, this being the first case reported in which the pleura was involved."

MARRUGAT, O. L. Hidatidosis muscular. [**Hydatids in Muscle.**] *Rev. Asoc. Méd. Argentina.* 1947, June 30, v. 61, No. 608, 486–7.

A report of 4 cases.

GALLIARD, H. Les types de développement exogène de *Strongyloïdes stercoralis*. Leur transformation par passages expérimentaux. [**Exogenous Developmental Forms of** *Strongyloides stercoralis*.] *C. R. Soc. Biol.* 1947, Feb., v. 141, Nos. 3/4, 102–5.

In the exogenous phase of *Strongyloides stercoralis*, there are alternative courses of development; in the direct—asexual or homogonic—cycle, the filariform larva develops directly from the rhabditoid larva passed in the faeces, and in the indirect—sexual or heterogonic—there is an intermediate development of male and female rhabditoides. An individual may be infected exclusively with worms showing one or other type of development, or with worms showing both types of development. It has been claimed by some workers that the indirect type of development occurs in the tropics and the direct in temperate climates, but other workers have shown that the direct development, either pure or mixed with the indirect type, also occurs in the tropics.

Working in Indo-China with the local strain and with strains from North Africa, the author has shown that, although the direct type of exogenous

evolution predominates in human infestations of *S. stercoralis* in temperate climates, this type also exists in tropical countries and particularly in Indo-China.

In Indo-China there is a determining factor which causes the strains of *S. stercoralis* of human or canine origin always to develop into the same evolutionary type, *i.e.* the mixed type with the indirect type predominating, very rarely the indirect type alone. This state of stability is reached after a variable number of passages with the dog as host, and it persists indefinitely (88 passages in 5 years in one case, 27 in another, 34 passages in one canine strain, all voluntarily interrupted). This confirms the author's observations on the extreme sensitivity of dog to the human parasite in that country [this *Bulletin*, 1940, v. 37, 650].

This tendency is not conditioned by the nature of the strongyloides larvae as was shown by the results of using separate evolutionary types of larvae, direct, indirect or mixed.

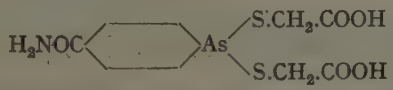
There is no correlation between the exogenous evolutionary type of larvae in man, or its transformation by passage in the dog, and the virulence of the strain. Contrary to Faust's claim that transformation in a given type is accompanied by a diminution in the virulence which in the long run makes the human strain inadaptable to dogs, the author's strains, which have developed into an almost pure indirect evolutionary type, have not lost their virulence after a very large number of passages. *L. E. Napier.*

HU, S. M. K. **Notes on the Experimental Infection of** *Culex pipiens* **var.** *pallens* **Coq. with** *Microfilaria malayi* **Brug.** *Chinese Med. J.* Shanghai Edition. 1946, July–Aug., v. 64, Nos. 7/8, 213–17.

"These infection experiments confirm previous findings in Shanghai indicating the low susceptibility of *Culex pipiens* var. *pallens* to infection with *Microfilaria malayi*, even when there were high microfilarial counts in the blood of the patient at the time of infecting the mosquitoes."

OTTO, G. F. & MAREN, T. H. **Filaricidal Activity of Substituted Phenyl Arsenoxides.** *Science.* 1947, Aug. 1, 105–7.

Although antimony compounds have proved the most successful in the treatment of filariasis, the authors, in exploring this field, had no success against the adult forms of *L. carinii* in the cotton rat or of *D. immitis* in the dog. The activity of arsenicals against filarial parasites has been indicated previously [this *Bulletin*, 1941, v. 38, 151 ; 1945, v. 42, 304, 837]. Mapharsen has now been found to be very effective *in vitro*. A number of other phenyl arsenoxides were therefore investigated. On studying their effect against the microfilariae of the cotton rat and dog *in vitro* the author found that those with *p*-$CONR_2$ and *p*-$SONR_2$ groups showed greatest activity. Mapharsen itself was inactive *in vivo*, but other compounds of the series showed greater promise, and the dithioglycollate of p-arsenosobenzamide—



in suitable doses killed all the adult worms of cotton rats and dogs, while failing to eradicate completely the microfilariae in both of these animals. Experiments are now in progress with this substance in the treatment of filariasis in man.

J. D. Fulton.

GARNHAM, P. C. C. & MCMAHON, J. P. **The Eradication of *Simulium neavei*, Roubaud, from an Onchocerciasis Area in Kenya Colony.** *Bull. Entom. Res.* 1947, Mar., v. 37, Pt. 4, 619–28, 3 figs.

There are three onchocerciasis areas in Kenya, where the disease is transmitted by *Simulium neavei*. One of these, Kodera, is well isolated from other infested areas, is heavily infested, and has been studied in past years so there is a basis of knowledge of the seasonal prevalence of the vector, and was thus suitable for an experiment on the eradication of *Simulium neavei*.

The chief breeding-places are two rivers which ultimately unite, in which there is perennial breeding limited to well-defined stretches totalling 15 miles in length. Seasonal tributaries act as subsidiary breeding areas. The principles of the experiment were to apply a DDT/oil/water emulsion to the running water at the upper limit of the infested streams in sufficient quantity to maintain a concentration of at least 2 parts DDT per million parts of water for 30 minutes, repeating the process once every 10 days in order to kill successive batches of larvae, and checking the results by searches for larvae and pupae, but more particularly by searches for adult flies.

Various emulsions were used. The most successful was 20 per cent. DDT in toluene, with 10 per cent. soap and 1 per cent. "Abracol" as a stabilizer, diluted to an unstated extent with water before use. The quantities necessary were calculated by accurate measurements of the rate of flow of the rivers at the lower end of the portion to be treated. Apparently each river was dosed at one point only above the upper limit of infestation, the lengths to be treated being six and nine miles. The emulsion was applied from drip cans, previously calibrated, suspended over the water. The first six applications were at 10-day intervals, the next five at roughly 14-day intervals, and the last after a month, the total period covered being five months. Tributaries were treated on four occasions during the rains.

Larvae and pupae rapidly disappeared except at one point below a long stagnant stretch of river, where they persisted until the arrival of the rains produced a good flow, and larvae finally disappeared three months after the start. Adult flies could no longer be caught one month after the start, though intensive searches continued for seven months longer. Experience of previous years, and of other areas in Kenya, makes it very unlikely that this was a seasonal decline, and the opinion is expressed that complete eradication of *Simulium neavei* has been secured.

Fish were killed in some numbers, but this was thought to be a relatively minor nuisance. Other insect life decreased but subsequently became re-established. *Simulium alcocki*, which is not a vector of onchocerciasis, reappeared after the end of the experiment, but it was known to have been breeding above the points where the rivers were treated, and thus to be in a ready position to infiltrate again into the treated zone. The nearest area infested by *S. neavei* being six miles away and separated by a ridge of hills, reinfestation is thought to be unlikely. *G. Macdonald.*

CASIS SACRE, G. Estado de nuestros conocimientos, sobre la oncocercosis. [**Present Knowledge regarding Onchocerciasis.**] *Medicina.* Mexico. 1947, Aug. 10, v. 27, No. 537, 345–50.

An examination of the present position and future requirements.

DEFICIENCY DISEASES.

PIERS, F. **Notes on some Cutaneous Manifestations of Deficiency Disease.** *East African Med. J.* 1947, July, v. 24, No. 7, 246–58. [23 refs.]

Twenty-two cases of a deficiency syndrome occurred in a large mental hospital. The outstanding features were those of classical pellagra, but most patients exhibited signs, mainly cutaneous, of deficiency of other B-complex factors, vitamin A, and possibly amino-acids. The interrelationship of these manifestations and the influence of environmental factors other than nutrition in their production are discussed at length.

[The presence of a number of inaccuracies and inconsistencies lessens the confidence with which the conclusions can be accepted, *e.g.* on p. 247 " The remainder (*of the calories*) was made up by vegetables (mainly roots)," whereas in Table I—" Vegetables—greens—present amount—8 oz.", and on p. 250, " The follicles are . . . plugged by greasy masses of inspissated horny detritus."]

Dean A. Smith.

VAN VEEN, A. G. & POSTMUS, S. **Vitamin A Deficiencies in the Netherlands East Indies.** *J. Amer. Dietetic Ass.* 1947, Aug., v. 23, No. 8, 669–73.

This paper summarizes the results of a series of clinical and dietary surveys, particularly in respect of vitamin A status, undertaken in 1937–39 by the Eijkman Institute and Institute for Nutrition Research, Batavia.

Evidence of vitamin A deficiency—xerosis conjunctivae, xerophthalmia and impaired dark adaptation—was found almost entirely in children and more commonly in boys than girls. The dietary survey revealed that the consumption of vegetables is very small in children, especially boys, and that of animal food negligible. Moreover, it was in the more prosperous areas, where least vegetables are eaten, that the incidence of deficiencies was highest.

Red palm oil proved effective in treatment, given either by mouth or intramuscular injection.

A microchromatographic method was used to estimate the individual carotenoids in food and serum. It was found among Javanese prisoners that, even on a low carotene intake, serum vitamin A values were high and values for α- and β-carotene low, indicating a high efficiency of conversion of these provitamins. On the other hand, serum cryptoxanthine levels were high and the authors conclude that this carotenoid does not serve as a provitamin A in human beings as it is known to do in rats. *Dean A. Smith.*

SPRUE.

BLACK, D. A. K., BOUND, J. P. & FOURMAN, L. P. R. **Fat Absorption in Tropical Sprue.** *Quart. J. Med.* 1947, July, N.S. v. 16, No. 63, 99–109. [15 refs.]

Impairment in fat absorption as shown by the excretion of fatty stools is the most constant and characteristic feature of the sprue syndrome ; but simple estimation of the percentage of fat in the dry stool, does not give quantitative information on the amount of fat actually absorbed, since it takes no account of the amount of fat in the diet, nor does it measure the total excretion over

any known period. When, however, a sprue patient is placed on a controlled diet of known fat content, and all the stools for a definite period are collected, weighed and analysed, it then becomes possible to correlate fat intake to fat output. The percentage of fat absorbed over the period in question can be expressed as :—

$$\frac{(\text{dietary fat}-\text{faecal fat})}{\text{dietary fat}} \times 100$$

Fat-balance studies have been made on these lines on idiopathic steatorrhoea, but not in tropical sprue.

The material for study consisted of 28 soldiers who had acquired sprue on tropical service in India. All had steatorrhoea, and had lost 10 per cent. of body weight, whilst glossitis, moderate anaemia and abdominal distension were common. Two standard diets were used—one with 65 to 70 gm. of fat per diem, the other with 95–100 gm. Two main difficulties in interpretation were encountered, one, the unexpected variability of fat output, and the other the occasional occurrence of unpredictable spontaneous improvement. Duplicate estimations showed that the error of estimation of fat in the dried stool was less than 5 per cent., but it was found that successive four-day periods might differ in their apparent fat output by as much as 50 per cent. The chief method of overcoming this variability over short periods was to use periods of adequate length, that is, to group three periods of four days into a single 12-day period. Spontaneous improvement is not uncommon in patients who are at rest in bed on a controlled diet. Difficulties in interpretation arose from occasional incidence of watery diarrhoea and in some from the change of a 65 gm. to a 96 gm. fat dietary, and from the possibility that some part of the faecal fat is a true excretion and not unabsorbed food-fat ; in five patients, however, such a change did not affect the interpretation of fat balance results.

On a fat-free diet, patients suffering from sprue excreted no more fat than normal subjects.

In 19 patients, the percentage of fat absorption varied from 51 to 85 with a median of 75·6 and a median of 80, whilst in normals, fat absorption exceeds 90 per cent. of the intake. Excreted fat forms a smaller proportion of the faecal fat in the sprue stool, in which unabsorbed food fat is much higher than in normal persons. A 10 per cent. failure in fat absorption suffices to double the normal daily output of fat in the stools. From the point of view of treatment, the wide variation between fat absorption in different patients is of less importance than the changes which a single patient is likely to show from one period to the next, without change in treatment.

Fat absorption figures in two patients treated with nicotinic acid 50 mgm. thrice daily, and in four with the same dose, together with riboflavin 5 mgm. daily—both intramuscularly—showed no individual change in fat absorption, but taking the series as a whole there was, in five out of six, a small drop in fat absorption. No clinical improvement was observed with either nicotinic acid or riboflavin ; on the other hand, patients who were kept on liver treatment for a month or longer showed a gradual improvement in fat absorption. The striking feature is that with doses of liver extract sufficient to produce rapid clinical improvement, there is such a small and gradual effect on the fat absorption defect.

The liver preparations were " T.C.F.", an Indian preparation, containing most of the B-complex substances present in the original liver ; " Hepastab " (Boots) a moderately refined extract ; and " Hepatex " (Evans), an extract containing the whole of the vitamin B complex naturally present in liver with added thiamine and nicotinic acid. The standard dose was 4 cc. daily for all preparations and in all a loading dose of 40 cc. spread over four days, was given

intramuscularly. Yeast extract, "Vegemite", resembling "Marmite" was given in a dose of 5 gm. four times daily. In all cases this was combined with 4 cc. or 2 cc. of liver extract daily.

In individual patients, the variance in the fat excretion over a three- or four-day period was significantly smaller when the patients were having yeast extract.

Many patients with early sprue had a normal chylomicron count. In explaining the main results, the authors incline to the view that the action of yeast is a dose-effect and the large amount of yeast contains more of a substance active in improving fat absorption in sprue, whilst liver almost certainly contains some of this substance. Folic acid improves the anaemia of sprue and the diarrhoea and probably fat absorption as well. It is probable that the active principle in yeast extract is also due to folic acid. *P. Manson-Bahr.*

FOURMAN, L. P. R. **Changes in Blood Phosphate after Ingestion of Glucose and Fructose in Sprue.** *Brit. Med. J.* 1947, Sept. 13, 411–13, 2 graphs. [10 refs.]

When glucose is given by the mouth to normal subjects there is a fall in the plasma inorganic phosphate—a fall which is related to the metabolism of glucose. The changes in serum inorganic phosphate during glucose absorption in sprue have already been investigated by HANES & REISER [this *Bulletin*, 1941, v. 38, 389] who found an abnormally small drop in serum inorganic phosphate in sprue when glucose was given by the mouth, but the normal drop occurred when it was given intravenously: but they found also that the urinary excretion of phosphate after glucose ingestion is greater in patients suffering from sprue than in normal persons.

Five normal persons and four sprue patients were studied. All the latter had steatorrhoea and had lost weight. In each, observations were made after the ingestion of glucose (50 gm.) and fructose (50 gm.).

The following data were obtained for each experiment: (1) Plasma inorganic phosphate; (2) whole-blood inorganic phosphate, by means of the Bodansky (1933) method; (3) fasting and hourly urinary excretion of inorganic phosphate up to two hours, by the Briggs (1922) method; and (4) packed cell volume.

Data were also obtained in three sprue patients and three normal persons on the "ester" phosphate changes, by means of Briggs's method. The results in normal persons showed that fructose ingestion is followed by blood-phosphate changes similar to those observed after taking glucose. Using the fall in the whole-blood inorganic phosphate as evidence that the sugars have been absorbed, the author found that two sprue patients had some impairment in the absorption of both glucose and fructose. Like glucose, fructose can form phosphate esters, and a defect in phosphorylation, which may be present in sprue, would be expected to interfere with the absorption of both these sugars.

P. Manson-Bahr.

ADLERSBERG, D. & SCHEIN, J. **Clinical and Pathologic Studies in Sprue.** [Abridged.] *J. Amer. Med. Ass.* 1947, Aug. 23, v. 134, No. 17, 1459–67.

This report concerns itself with clinical and pathological observations in a group of 40 patients observed at the Mount Sinai Hospital during the past fifteen years. Thirty-six of them were classified as "primary sprue". In six of these latter, post-mortem examinations confirmed the absence of gross systemic disease, although alterations were found in the intestinal canal and

mesenteric glands. In four of the secondary group, gross pathological changes were encountered which could be held responsible. The group with primary sprue comprised 36 patients, 13 men and 23 women. Of these, 15 were Latin-Americans and the remainder first-generation Americans of Central and Eastern European extraction, but there was a preponderance of Jews and Porto Ricans. In age they varied from 16–73, the average being 41. In this series, 7 contracted their illness in the tropics, whilst 29 acquired the disease under American climatic conditions. Observations were made over the course of one month to twelve years. The average was 3·6 years. The results of investigations on the biochemical and haematological aspects do not differ from the generally accepted findings, but the conclusions regarding vitamin A and fat tolerance are of some interest. The former was studied in 15 cases. In 12, during the active stage, the fasting vitamin A level ranged between 15 and 63 microgrammes (the average being 35 microgrammes per 100 cc.). This represented a reduction of about 50 per cent.

The disturbance in vitamin A metabolism is characterized by low fasting figures and lack of elevation in the post-absorptive phase and is paralleled by the low carotene levels of the serum. Three patients during remission showed that this disturbance may persist in a modified form.

An analogy may be drawn between this observation and the persisting steatorrhoea and the persisting X-ray abnormalities of the small intestine.

In assessing the effects of therapy, a classification based on a 1 plus to 4 plus change in the reticulocytes, haemoglobin and red blood corpuscles disclosed that 6 patients had a 4 plus response, 14 a plus 3 response and 9 a 1 plus response. Fatalities, however, occurred even in those with good original responses of 1 to 3 plus.

Although, in general terms, the initial haematological response was not so striking as the coprological, nevertheless in terms of return to normal, the elevation of the blood count superseded the intestinal improvement.

As regards increase of weight, sense of well-being and alleviation of glossitis, 27 patients showed remarkable response to specific therapy and 3 none at all. There were 8 deaths, of which 6 came to necropsy. Of the four secondary sprue patients, the duration of symptoms ranged from three to six months, prior to hospitalization. Clubbing of fingers was observed in one. Response to liver therapy was uniformly poor. All four patients in this group died.

In the secondary group, massive involvement of the mesenteric glands interfered with absorption.

Haematological observations differed in the two groups. In secondary sprue, the haemoglobin and erythrocytes were on a higher level than in primary sprue. The colour index is of differential diagnostic aid. In 89 per cent. of cases of primary sprue, the colour index was 1 or less than 1. In secondary sprue, it was invariably less than 1. The leucocyte count averaged 6,712 in primary and 10,525 in secondary sprue.

The typical oral and lingual lesions of primary sprue apparently require many months or years for their full development and are of minor importance in secondary sprue.

Haemofuscinosis of the muscularis propria of the gastro-intestinal tract was present in two patients. It is probably related to the "ceroid" pigment described by PAPPENHEIMER and VICTOR [*Amer. J. Path.*, 1946, v. 22, 395] in animals deficient in vitamin E and in man with malabsorption syndromes.

Hyaline lesions at the tips of the villi of the small intestine are described in one case. No characteristic changes were seen in the large intestine.

In secondary sprue, amyloid deposition in the small intestine was described in two cases. The significance of this finding is not apparent and its importance has not received sufficient attention. *P. Manson-Bahr.*

SUÁREZ, R. M., SPIES, T. D. & SUÁREZ, R. M., Jr. **The Use of Folic Acid in Sprue.** *Ann. Intern. Med.* 1947, May, v. 26, No. 5, 643–77, 15 figs. [15 refs.]

This report is based on the study of 50 cases of sprue, of which 22 were in the acute stage. The object of the investigation was to determine the optimum oral dose of folic acid and its effect on patients given a diet inadequate in meat, fish and eggs, and from which liver and yeast were excluded ; and also its effect in the presence of an adequate diet, high in animal protein, but low in fats and carbohydrates. The influence of this dose upon the glucose tolerance curve and upon the fat content of the stools was examined ; and the maintenance dose of folic acid was determined. The question whether folic acid can be substituted for parenteral liver extract in the treatment of chronic cases had also to be decided.

The paper is replete with protocols of cases, and records of the detailed and prolonged investigations on the biochemical and haematological responses in each patient, which it is impossible to summarize. There is also a pathological section based upon 16 autopsies, which is equally detailed, but which does not add materially to existing knowledge of this subject. The shortening and blunting of the intestinal villi of the small intestine in half this number was noted.

The efficacy of folic acid in the treatment of tropical sprue is confirmed. Oral administration of 10 mgm. daily is adequate, and it is found that small daily doses are more effective than 50 times as much in a single dose. Observations on five cases suggest that a daily dose of 20 mgm., together with an adequate sprue diet produces better results than larger doses accompanied by an inadequate diet. The impression is obtained that from 2·5 to 5 mgm. is an adequate maintenance dose in most cases. For patients on a diet high in animal proteins and low in carbohydrates and fat, a smaller amount of folic acid is necessary than for those on a diet low in animal protein but high in carbohydrate and fat.

In chronic cases of sprue, injections are given of Lilly's crude liver extract solution of 2 injectable U.S.P. units per cc. (containing approximately 4·21 micrograms of folic acid per cc.). Of this the authors have been using 6 cc. daily intramuscularly.

It is suggested that liver extract contains some other anti-anaemic substance independent of, and unrelated to, folic acid.

Although it is not precisely stated, it appears that this course is necessary in order to obtain a haematopoietic response. *P. Manson-Bahr.*

HAEMATOLOGY.

RAO, M. N. & KARMAKAR, G. **A Note on estimating Haemoglobin with One Cubic Millimetre of Blood.** *Indian Med. Gaz.* 1947, Mar., v. 82, No. 3, 120–22.

The usual methods of estimating haemoglobin require a minimum of 20 cmm. of blood. With small animals it is sometimes difficult to obtain this amount. For a total red cell count, only 1 cmm. is required. The authors have devised a method of estimating the haemoglobin with the balance of the fluid after the red cell count has been made, by adapting the pseudo-peroxidase reaction for detecting minute quantities of blood.

The requirements are (i) a standard 1 per cent. benzidine solution, and (ii) a standard blood solution. (i) Commercial benzidine, although marked " pure", always gives a coloured solution and must first be purified to reduce the colour to a minimum : a slight residual colour can be allowed. (ii) The blood solution is made in 1 per cent. boric acid from a blood of known haemoglobin value.

In each of three test tubes, 1 ml. of benzidine solution is placed. To the first tube, 0·5 ml. of standard blood solution, to the second, 0·5 ml. of the unknown blood, and to the third, distilled water are added. To each tube 0·5 ml. of 1 to 2 per cent. hydrogen peroxide is added, and the colour deepens from green to purple in 2 hours. To each, suitable but equal amounts of 20 per cent. acetic acid are now added. The cells in the Pulfrich photometer used for matching are 0·5 ml. thick and No. 11 filter is used. The standard and the unknown are successively matched, with the control in the other cell in each case. The density drum corresponding to the cell filled with the blood solution is always set at zero, and the other drum containing the control solution is rotated in order to find the extinction co-efficient. The result is calculated :

$$\text{Hb. content of unknown} = \frac{\text{extinction coefficient of unknown}}{\text{extinction coefficient of known}} \times \text{Hb. content of known}$$

In practice, once the extinction coefficient of the known is estimated:

$$\frac{\text{Hb content of known}}{\text{extinction coefficient of known}} \text{ becomes a constant}$$

and the haemoglobin content of the unknown can be calculated simply by multiplying the extinction coefficient of the unknown by this constant.

Two series of tests by the Pulfrich photometer, with the Hellige method as the control, were carried out ; in the first (14 samples), commercially pure benzidine and in the second (10 samples) specially purified benzidine were used. The correlation coefficient between the results of the two methods was over 0·9 in each case. It is concluded that the method is sufficiently accurate for research purposes, but that for this the purified benzidine should be used. For routine purposes commercially pure benzidine could be used.

L. E. Napier.

PIJPER, A. **The Diffraction Method of measuring Red Blood Cells.** *J. Lab. & Clin. Med.* 1947, July, v. 32, No. 7, 857–77, 50 figs. (4 coloured). [50 refs.]

A chance observation of the diffraction of light through a bacterial culture led the author to study the estimation of bacterial size and later of erythrocyte diameters by means of diffraction. A parallel beam of light, produced with or without a condenser from a small bulb in the bottom of a box, is passed through the thin portion of an evenly spread blood film, and the resulting circular spectrum produced by diffraction of the light by the blood cells is projected through a simple convex lens on to a ground-glass screen ; in some instruments this lens is placed between the light source and the blood film. The inner complete spectrum, which extends from an inner violet ring to an outer red ring, is used for the estimations, the coloured rings of incomplete spectra outside this being disregarded. The variously sized cells superimpose their respective spectral rings, but the final composite position of the yellow ring in the complete spectral ring is hardly affected by the superimposition of the diffraction patterns of all the red cells. The yellow ring, which forms a narrow band easy to read and sufficiently near to the middle of the spectrum to be suitable, is used to estimate the mean cell diameter, while the outer edge of the red circle is an expression

of the diameter of the smallest cells in the film and the innermost violet circle is an expression of the largest cells. Thus not only can the mean corpuscular diameter be estimated but also the maximum and minimum cell diameters, while a consideration of the spread of the spectrum reveals the degree and kind of anisocytosis. Poikilocytosis causes a watery ill-defined spectrum difficult to measure, but this in itself is useful information. The author uses a lens of 20 cm. focal length, and it can be shown that, to an accuracy of about 1 per cent., the diameter of the cells is $\frac{340{,}000\lambda}{r}$ where r is the radius of the spectral ring on the ground glass and λ is the wavelength of the light; the radii of the rings range from about 1·5 to 3·5 cm. and may be measured by means of a pair of dividers. In a refinement of the apparatus, two equal beams of light are passed, one through a normal blood film and the other through the blood film to be examined. Their spectral rings are then projected by the lens concentrically on to the ground-glass screen, but reciprocal halves of each pattern are blanked off by an axially placed opaque plate. Thus each film produces half a spectral circle and the two halves meeting along a diameter render comparison of sizes and therefore any change in the test film from the normal immediately visible. The author claims the diffraction method to be free of many errors associated with other methods of estimating erythrocyte diameters and more practicable than determinations by means of Price-Jones curves. *F. Murgatroyd.*

ALTMANN, A. **The Survival of Transfused Erythrocytes in Sickle-Cell Anaemia.** *Trans. Roy. Soc. Trop. Med. & Hyg.* 1947, July, v. 40, No. 6, 901–4, 1 chart. [16 refs.]

"Normal cells transfused into a patient with active sickle-cell anaemia showed normal survival with a linear disappearance curve. The haemolytic process in sickle-cell anaemia only affects the patient's own red cells which are abnormal due to a hereditary factor, whilst normal red cells remain unaffected."

BEET, E. A. **Sickle Cell Disease in Northern Rhodesia.** *East African Med. J.* 1947, June, v. 24, No. 6, 212–22. [16 refs.]

Of 1,289 Africans examined in the Eastern Province of Northern Rhodesia, 154 showed sickling of their red cells, giving an incidence of 11·9 per cent., which compares closely with the incidence of 12·9 per cent. obtained with Bantu of the Balovale District in Northern Rhodesia [see this *Bulletin*, 1946, v. 43, 774].

No significant difference in incidence due to sex, tribe, malaria, schistosomiasis or ancylostomiasis could be determined. In young children, the incidence appeared higher than in older persons, suggesting that more of those with sicklaemia die before the age of about five years than do normal children, possibly because the former are more liable to contract intercurrent disease. As the spleen rate for those without sicklaemia is higher than that for those with the trait, it is suggested that the spleen in sicklaemia is liable to attacks of thrombosis. Sickle cell disease is often associated with a severe anaemia, but the anaemia is more frequently due to some intercurrent condition than to the sickle cell disease, although the latter may in itself at times cause anaemia.

F. Murgatroyd.

TRINCÃO, C. O mielograma na anemia de células falciformes. [**The Myelogram in Sickle-Cell Anaemia.**] *An. Inst. Med. Trop.* Lisbon. 1946, Dec., v. 3, 81–92, 1 fig. [19 refs.] English summary (7 lines).

A review and 4 cases.

SCOTT, Annie V. **Cooley's Anemia (Mediterranean Anemia) in a Chinese Child.** *Chinese Med. J.* Shanghai. 1947, Mar.–Apr., v. 65, Nos. 3/4, 77–84. [11 refs.]

DAVIDSON, L. S. P. **Advances in the Treatment of Blood Diseases.** *Practitioner.* 1947, Oct., v. 159, No. 952, 311–19. (17 refs.]

KEMP, T. A. **Liver and Folic Acid in the Treatment of Nutritional Macrocytic Anaemia. A Comparison of Results.** *Lancet.* 1947, Sept. 6, 350–53, 4 figs.

"In 3 cases of nutritional macrocytic anaemia with megaloblastic hyperplasia of the bone-marrow folic acid produced a more rapid improvement in the peripheral blood than would have been expected with large doses of liver extract parenterally and iron by mouth. The bone-marrow resumed a normoblastic pattern."

GRANIER-DOYEUX, M. & HOLZ, S. Factores antianemicos de reciente adquisición con especial mención del "Acido Fólico" y factores afines. [**New Anti-Anaemic Factors, with special reference to the "Folic Acid" Group.**] *Rev. Sanidad y Asistencia Social.* Caracas. 1946, June–Aug., v. 11, Nos. 3/4, 189–256, 1 fig. [233 refs.]

A comprehensive review of present knowledge.

WELCH, A. D. **The Present Status of Pteroylglutamic Acid and of other Hematopoietic Agents.** *Federation Proc.* Baltimore. 1947, June, v. 6, No. 2, 471–9. [40 refs.]

DERMATOLOGY AND FUNGUS DISEASES.

CURTIS, A. C. & GREKIN, J. N. **Histoplasmosis. A Review of the Cutaneous and Adjacent Mucous Membrane Manifestations with a Report of Three Cases.** [Abridged.] *J. Amer. Med. Ass.* 1947, Aug. 9, v. 134, No. 15, 1217–23, 7 figs. [Refs. in footnotes.]

This paper presents a useful analytical survey of the published reports of histoplasmosis relative to lesions on the skin and mucous membranes, and the authors add three new cases in which lesions on these tissues were a salient clinical feature.

In case 1 (a white woman aged 30), the visible signs of disease were disseminated, subcutaneous nodules which tended to undergo caseation-necrosis, break down and ulcerate on the skin surface, discharging a thin yellowish fluid. A nodule in the cervical region enlarged to the size of a hen's egg, but other nodules did not exceed a few centimetres in diameter. There was no significant involvement of the lymph nodes. Some of the lesions healed after a few months, but others persisted. Constitutional symptoms, with fever and night sweats, were marked, and there was evidence of a generalized disease in which the lungs were extensively involved. Repeated, direct examinations

of material from the skin lesions failed to reveal the nature of the disease, but the diagnosis of histoplasmosis was made eventually by the discovery of *Histoplasma capsulatum* in the sputum. The case ended fatally.

Case 2 (a male negro aged 42), suffered from a fungating ulcer measuring 5·0 by 2·5 cm. on the left side of the tongue, associated with swelling and tenderness of that organ, but no restriction of its movements, and accompanied by crusted lesions on the chin and nares. Some of the cervical lymph nodes were swollen and painful and, after incision, discharged a sanguineous material, intermittently, for about four months. By microscopic examination of sections from the tongue lesion, the condition was diagnosed as histoplasmosis, but, despite malaise, night sweats and loss of weight, there was no evidence of generalized infection. Treatment with sulphadiazine, 6 gm. by mouth daily up to a total dosage of 576 gm., resulted in " steady clinical remission " ; the cutaneous lesions cleared after several weeks and the tongue appeared to be quite normal in less than three months. Although the authors do not claim an unequivocal cure, the result of sulphadiazine treatment is very significant, as hitherto no drug, with the possible exception of antimony, has had any observed effect on this disease.

In case 3 (a male negro aged 25), the disease appeared to be confined to the prepuce and glans penis, with involvement of the inguinal lymph nodes on both sides. Very marked improvement resulted from the administration of sulphadiazine ; 522 gm. over a period of 87 days. *J. T. Duncan.*

FERNÁNDEZ LUNA, D., EMILIO CASET, I. & ABBATE, E. A. Nueva observación de paracoccidioideomicosis. (Forma buco-laringo-pulmonar). [**A New Case of Paracoccidioidomycosis (Buccal-Laryngeal-Pulmonary Form).**] *Rev. Asoc. Méd. Argentina.* 1947, Aug. 15, v. 61, No. 611, 571–5, 14 figs.

MARANO, A. & NIÑO, F. Localización ganglionar del *Paracoccidioides brasiliensis.* [**Ganglionic Localization of** *Paracoccidioides brasiliensis.*] *Rev. Asoc. Méd. Argentina.* 1947, Aug. 15, v. 61, No. 611, 570–71, 4 figs. English summary (7 lines).

SHAFFER, L. W. & ZACKHEIM, H. S. **Sporotrichosis. Report of a Case in which Treatment with Iontophoresis was Successful.** *Arch. Dermat. & Syph.* 1947, Aug., v. 56, No. 2, 244–7, 1 fig.

TROPICAL OPHTHALMOLOGY.

A REVIEW OF RECENT ARTICLES. XLIX*.

Photophthalmia.—Solar photophthalmia among a large number of motor-convoy drivers employed during the war in the Northern Territory of Australia is discussed by DWYER[1]. Each year from mid-September to April, which is the hot season, the incidence of conjunctivitis increased amongst these men. It was least in the months from April to September and after rainfall. The conjunctivitis came on after an interval, generally about midnight or in the early hours of the morning ; it was not purulent and no significant bacteria could be cultivated from the affected eyes. Intense photophobia, blepharospasm and lachrymation were present and were most severe on the first day.

* For the 48th of this Series see Vol. 44, pp. 611–618.

[1]DWYER, J. M. Solar Photophthalmia. *Med. J. Australia.* 1947, Apr. 26, v. 1, No. 17, 523–5, 1 fig.

The bulbar conjunctiva was intensely congested, but usually only in the areas not covered by the eyelids, although the tarsal conjunctiva was also to a lesser extent affected as well. Sometimes corneal involvement occurred in the form of superficial punctate lesions confined to a horizontal band across the cornea corresponding to the area bounded by contracted lids. At the end of 1942, the number of cases rose to nearly 200 a week.

Eye shields, goggles, dark glasses and eye shades were tried in an attempt to prevent this type of ophthalmia, but all were discarded either because they were uncomfortable or because they were of very little benefit. A green strip two inches wide was painted across the top of the windscreens of the motor vehicles for the purpose of obscuring the view of the sky. The men all commented on the sense of comfort the green shade gave them, and it proved to be most efficacious in preventing conjunctivitis.

The author considers that, as mere shading out of the sky controlled this solar ophthalmia, it is due to the absorption of rays direct from the sky and not from the earth's surface nor from the direct rays of the sun.

Blindness.—An analysis of the causes of blindness in 122 cases in Algiers is made by GAUTHIER[2]. Trachoma is responsible for the largest number and occurred in 30 of the cases. Smallpox is the cause in 22 cases. Syphilis, responsible for the loss of so many eyes in all countries, is only represented by 2 cases of interstitial keratitis, but is probably a factor in the causation of optic atrophy in 20 cases and infantile glaucoma in 6 cases. Purulent ophthalmia in infancy was the cause in 16 cases. The author points out that smallpox as a cause of blindness in Algiers must not be overlooked.

Nutritional Deficiency.—Nutritional retrobulbar neuritis in children in Jamaica is discussed by WHITBOURNE[3] and CARROLL[4]. The children were all of mixed European and African blood or of pure-blooded African stock, between the ages of 9 to 16 years; girls were more affected than boys. The vision varied from 6/9 to 2/60. The optic discs were normal in appearance early in the disease, but later often showed marked temporal pallor. The visual fields showed small definite central or centro-caecal scotomata. The peripheral fields were normal. Approximately 10 per cent. of the children had partial deafness and most of them showed changes in the skin or mucous membrane characteristic of vitamin B complex deficiency. These consisted of excoriations at the angles of the mouth and occasionally at the outer canthi, greasy desquamations along the nasolabial folds, sore mouths and sore tongues. No gross neurological signs were noticed, except those of the second and eighth nerves. Recovery depended on improvement in the patients' diet, or at least in adding yeast to the diet, provided that an early diagnosis was made and treatment started promptly. The condition appears to be similar to the syndrome found in numerous American soldiers released from Japanese prisoner-of-war camps.

Exposure to Atebrin [Mepacrine].—Blue haloes in atebrin workers are discussed by MANN[5]. Her observations are made on the eye condition of six workers engaged in the production of the drug and its compression into tablets. The patients were healthy men, had been working 9 hours a day for five days a week in an atmosphere which often contained for short periods

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[2]GAUTHIER, A. Statistique personnelle sur les cas de cécite vus en 1946 à Alger. *Algérie Méd.* 1947, Jan., No. 1, 80–81.

[3]WHITBOURNE, Dahlia. Nutritional Retrobulbar Neuritis in Children in Jamaica. *Amer. J. Ophthalm.* 1947, Feb., v. 30, No. 2, 169–71.

[4]CARROLL, F. D. Nutritional Retrobulbar Neuritis. *Amer. J. Ophthalm.* 1947, Feb., v. 30, No. 2, 172–6, 3 figs.

[5]MANN, Ida. "Blue Haloes" in Atebrin Workers. *Brit. J. Ophthalm.* 1947, Jan., v. 31, No. 1, 40–46, 4 coloured figs. on 1 pl.

clouds of fine atebrin dust. They wore protective clothing, goggles and masks, but the dust was so fine that in spite of this they soon noticed yellow staining of the conjunctivae and skin. After working for some weeks or months, they all noticed a blue halo round lights. These haloes were darker blue near the light and pale blue at its outer edge. If the light was placed further away at six metres, faint yellow, green and reddish brown bands appeared outside the blue ring. The visual acuity was unimpaired, there was no ocular pain, lachrymation or photophobia.

Examination with the corneal microscope showed a diffuse pale yellow stain of the conjunctiva in the interpalpebral space only; aggregations of minute dark brown spots were found at the limbus in the exposed portion and also under the edge of the lower lid, but not in the upper quarter of the limbus. They appeared to be an actual deposit, were not movable and could not be washed off.

The whole surface of the cornea was peppered with very fine dustlike particles, which appeared dark yellowish brown by direct illumination and opaque by transmitted light. As in the conjunctiva, the particles were intracellular or at least situated in the substance of the corneal epithelium. The substantia propria and the internal parts of the eye were normal. The particles are not composed of atebrin itself, since this is completely soluble in the tears, but of an insoluble derivative of it.

Complete recovery takes place on removal from contact with atebrin dust.

Optic Nerve Lesions in Typhus.—The frequency of lesions of the optic nerve in exanthematic typhus and murine typhus (flea typhus) is discussed by TOULANT and LARMANDE.[6] They point out that, although previous writers have reported that these lesions are rare, they have found that in 100 cases of exanthematic typhus bilateral optic neuritis occurred in 74. It occurs largely during the stage of meningeal involvement, that is between the seventh and twelfth day. They consider that the optic neuritis is not due to congestion or parenchymatous neuritis, but to a pure neuritis secondary to inflammation of the meninges; the extent of the optic nerve lesions is dependent upon the intensity of the typhus virus or its toxin. They found that the optic neuritis eventually cleared up completely with recovery of normal vision, but this recovery is slow and the ophthalmoscopic signs persist for one or two months. In a small number of cases, they found that the optic neuritis resulted in optic atrophy with vision more or less affected, even to the extent of blindness.

In murine typhus, they examined 14 cases and found optic nerve involvement in 9 cases. Of these 9 cases, 1 had hyperaemia of the optic disc, 4 mild optic neuritis, and 4 severe optic neuritis. *E. O'G. Kirwan.*

MULOCK HOUWER, A. W. Kerato-conjunctivitis epidemica. [**Epidemic Kerato-Conjunctivitis.**] *Med. Maandblad.* Batavia. 1947, Feb., No. 7, 120–23. [12 refs.]

OLLE, R. G. & HECTOR CAMBIASO, R. Zooparasitosis intestinal y ojos. [**Intestinal Parasitism and Ocular Disease.**] *Rev. Sanidad Milit.* Buenos Aires. 1946, Sept., v. 45, No. 9, 1055–76, 4 figs.

This is a paper which was read at an Ophthalmological Congress in the Argentine. The time has long passed, say the authors, when "eye diseases" constituted a separate and independent section of medicine and surgery. They

[6] TOULANT, P. & LARMANDE, A. Les lésions du nerf optique au cours du typhus exanthématique et du typhus murin. Leur fréquence. *Bull. Acad. Nat. Méd.* 1947, v. 131, Nos. 17/18, 334–6.

have examined the faeces of 67 patients presenting ocular symptoms and record their findings. The article is divided into four parts. In the first, brief notes are given of each of the 67 stating the ocular condition present—conjunctivitis, eczema of the lids, iritis, chalazion, keratitis, etc.—the results of faecal examination and the treatment adopted for the parasitism. The findings may be summarized : *Blastocystis* was found in 39, *E. histolytica* in 27, *Endolimax nana* in the same number, *E. coli* in 13, *Trichomonas* in 7, *Giardia lamblia* in 4, *Chilomastix mesnili* in 2, *Enterobius vermicularis* in one. In the second part, pathogeny is discussed shortly. The authors suggest [not very convincingly] that the presence of the parasite leads to dyspepsia, ariboflavinosis, toxaemia and allergy and so to the eye lesions and symptoms. This is again spoken of in Part 3, Symptomatology and Diagnosis ; and the authors recommend that all those with eye trouble who do not rapidly get well on the usual methods of treatment should be examined for intestinal parasites. [In a country where these are common, faecal examination should surely be a matter of routine, for it is well known that parasitism may delay the cure of any concomitant disease]. Part 4 deals with Prognosis and Treatment. Only 40 cases were followed up ; of these 16 were cured, 19 improved, 3 relapsed and 2 were refractory to treatment. The treatment was on the usual lines, and consisted of administration of emetine, amoebarsone, enterovioform and yatren or the parasitism, and, for the eyes, borax washes and lactoflavin, 3 tablets ? strength] daily. *H. Harold Scott.*

HEAT STROKE AND ALLIED CONDITIONS.

LEE, D. H. K. **Human Climatology and Tropical Settlement. The John Thomson Lecture for 1946.** 34 pp., 9 figs. [29 refs.] 1947. Brisbane : University of Queensland.

Opinions current twenty years ago were practically all adverse to the idea that a healthy virile productive white race could develop and continue unimpaired in a sub-tropical or tropical climate. However, CILENTO demonstrated that evidence was accumulating, which showed continuous adaptation to the environment (with regard to tropical settlement) in tropical Australia. The author refers to the confusion of thought and superficial reasoning underlying most statements made on the effects of tropical climates, and says that the major obstacle has been a totally inadequate appreciation of man's physiological adjustments to his environment. Out of this uncertainty has emerged the new science of human climatology.

The temperature-regulatory mechanism in man or the higher animals is very complex, and the determination of the thermal relationships between the human body and different environments therefore is difficult. But the central practical objective of human climatology is to determine the effect that a given climate may be expected to have upon the well-being of the individual. The difficulty is to discover a suitable index of well-being.

The multiple reactions of the body to hot enviroments are classified, and the more important factors affecting the degree and rate of production of these bodily reactions are listed. Over a large range of naturally-occurring climatic conditions, the amount of demonstrable physiological change is small, but there is evidence that efficiency and, it is said, initiative, deteriorate under conditions not sufficiently strenuous to provoke frank physiological reactions. It has been suggested that some difficulties occurring in laboratory studies might be overcome by careful observation over a period of time by trained workers living in a tropical community.

During the late war, considerable impetus was given to the study of clothing, but little attention was paid to housing. Any housing studies seem to have been directed towards protection from cold or the provision of full scale air-conditioning. House design for tropical regions has not advanced since 1925, and although many modern developments in design may be applied to tropical housing, many special problems remain. The problem of tropical settlement cannot be solved completely without the assistance of the social scientist.

In the past, population centres have grown up in the tropics almost entirely as dictated by commercial interests, but in the future, although commercial expediency will affect the location of commercial centres, physiological considerations should decide the precise site. Increased speed of communications and transport mean that administration need not necessarily be located in a commercial centre. Physiological considerations should also be taken into account in house design and town-planning.

Notwithstanding improved conditions, there will be few places in the tropics where absolutely unbroken residence will be permissible. Yet tropical residents must be *residents* and not mere *sojourners*, and a relief period must be one in which the person is going *away* from home, and not back home. A stable, contented, efficient white population cannot be established unless this fundamental principle is firmly established in practice. Detailed recommendations are made concerning the number and duration of long and short holidays in cooler climates, and interim recommendations are made regarding permissible conditions in hot industries in Australia. *F. A. Chrenko.*

LADELL, W. S. S. **Creatinine Losses in the Sweat during Work in Hot Humid Environments.** *J. Physiology.* 1947, July 31, v. 106, No. 3, 237–44, 3 figs. [10 refs.]

" 1. The creatinine contents of serial samples of sweat from men working in a hot humid environment were measured and compared with the creatinine contents of saliva and of plasma obtained at the same time.

" 2. Increasing the creatinine content of the blood raised that of the saliva correspondingly, but had little effect on that of the sweat.

" 3. There is a reciprocal relationship between sweat rate and the creatinine content of sweat."

MALAMUD, N., HAYMAKER, W. & CUSTER, R. P. **Heat Stroke. A Clinico-Pathologic Study of 125 Fatal Cases.** *Milit. Surgeon.* 1946, Nov., v. 99, No. 5, 397–449, 58 figs. (5 coloured). [71 refs.]

This paper gives a most comprehensive account of the clinical records and autopsy protocols of 125 cases of heat-stroke which occurred in members of the U.S. Army in the southern States of the U.S.A. in the summer months of 1941–45. Nearly all the cases occurred in persons undergoing heavy muscular work in high environmental temperatures. The authors distinguish clearly between the signs and symptoms due to the hyperthermia and those due to secondary shock and complications, including lower nephron nephrosis (renal anoxia). Three types of clinical course were observed : " (1) acute onset with early persistent coma and delirium ; (2) acute onset of early coma or delirium, but with remission and late relapse ; and (3) insidious onset with progressive course and late development of coma." The clinical picture depended to some extent on the duration of the illness. Laboratory findings included early leucocytosis, normal haemo-concentration, some increase in non protein nitrogen content of the blood and a decrease in CO_2 combining power. There was a

persistent early decrease of platelets and commonly a subsequent increase in prothrombin and bleeding times. Albumin, casts and red cells appeared in the urine in some case.

A first-class and beautifully illustrated account of the pathological changes is given. The changes in the central nervous system consisted essentially of progressive degeneration of neurons and replacement with glia. These changes were prominent in the cerebellum, cerebral cortex and basal ganglia, but not in the hypothalamus. The severity depended on the length of survival following the appearance of hyperthermia. Congestion and petechial haemorrhages and sometimes oedema were inconstant findings. The authors concluded that the cellular changes resulted from the excessive heat and the vascular changes from shock. Haemorrhages and parenchymal lesions were observed in other organs. Most of the lesions in organs other than the brain could be attributed to anoxia and circulatory failure consequent on the appearance of shock. Parenchymal lesions were most prominent in those who survived more than 24 hours; they included degeneration of megakaryocytes in the bone marrow (attributed by the authors to the hyperthermia), necrosis of heart muscle, lower nephron nephrosis and centrolobular liver necrosis.

The authors state that "the clinical, laboratory and pathological findings indicate that two factors are operative in heat-stroke: hyperthermia and shock." The changes in nervous tissue are primarily due to hyperthermia and not to anoxia and shock, in which the distribution of the brain lesions is different.

A very complete list of references is appended to the paper, which must be considered one of the best yet written on this difficult subject.

B. G. Maegraith.

MISCELLANEOUS DISEASES.

DAVIES, J. N. P. **Pathology of Central African Natives. Mulago Hospital Post Mortem Studies I and II.** *East African Med. J.* 1947, May & June, v. 24, Nos. 5 & 6, 180–84; 223–9.

These papers are based on the findings in 2,994 autopsies recorded at Mulago, Uganda, from May 1931 to May 1946. The examinations were made by a succession of medical officers [many of whom probably had had no special training in pathology] and in the earlier cases records are often inadequate and sections which may have been cut are missing. In 1,718 instances, diagnosis was macroscopic only.

Cancer accounted for 135 cases, syphilis 232, tuberculosis 354, pneumonias 710, meningitis, peritonitis, and septicaemia 388. As the author points out, none of the conclusions reached must be accepted more than temporarily until confirmed or otherwise by further work. The staphylococcus, pneumococcus, and streptococcus are all important causes of disease, the last named less so than in Europe, but primary pneumococcal lesions are commoner.

The most interesting part of the papers is that dealing with pyogenic diseases (II)—as the author quotes: "The commonest disease of Africans is pus." Meningitis, septicaemia, and pyaemia are discussed in some detail, and of the pyogenic organisms the pneumococcus is outstanding; out of 104 cases of primary meningitis this organism was responsible for 58, the meningococcus for 29. Thus the commonest endemic meningitis in and around Kampala is due to the pneumococcus, the mortality being a high one.

Sulphonamides were first used at Mulago in 1938, sulphapyridine was introduced in the same year but was not in general use till 1940, while penicillin was available in 1943, but is still not extensively used.

Pyogenic meningitis accounts for 4·4 per cent. of all autopsies. In acute bacterial endocarditis, the pneumococcus was responsible for 26 out of 76 cases and in purulent pericarditis 23 out of 46. The author has made out a good case for the importance of this organism to the African.

Twenty-four cases of systemic pyaemia were autopsied, and bacteriological examination made in 8 instances, all showing staphylococci. Pyomyositis accounted for 19 deaths, of which 11 were examined bacteriologically, 7 showing staphylococci, 2 streptococci and two pneumococci. As the author and others note the reason for the frequency of this condition in the tropics still remains a problem [see this *Bulletin*, 1947, v. 44, 751, 752].

Finally, it is remarked that to a large extent the pneumococcus is to the African what the streptococcus is to the European and is the first organism to be considered in cases of septicaemia.

[Two most interesting and thoughtful papers, which would well repay reading by all those practising in Tropical Africa. As the author rightly points out, any successful attempt to raise the general health of the African depends mainly on a greater supply of African doctors ; and while these have plenty of European text-books available, too little original work on the biology and physiology of the indigenous population is available. There is a great need for text-books written by those having long and wide experience of ordinary medical practice in the Tropics.

In conclusion it is to be noted that patients coming to Mulago include a high proportion of homeless, destitute and alien Africans.] *C. F. Shelton.*

WHARTON, H. J. & DUESSELMANN, W. **Favism. A Short Review and Report of a Case.** *New England J. of Med.* 1947, June 26, v. 236, No. 26, 974–7. [19 refs.]

A report of a case of favism in an American-born child of Italian extraction. The patient was a boy aged 3½ years. On admission, 16 hours after the onset of haemoglobinuria, he appeared to be moribund, temperature 102°F., pulse 160 per minute, red cell count 1·14 million cells per cmm., white cell count 20·5 thousand cells per cmm., platelets 248 thousand per cmm. ; no spherocytes or sickling observed. Urine contained blood pigments, albumin and coarse granular casts. There was no history of exposure to malaria, but the patient had been given a meal of fava beans on the day of the onset of the illness. Whole citrated blood was administered daily for 5 days with great clinical improvement. The patient was discharged eight days after admission. The spleen was not palpable on admission, but became so on the third day. It was no longer palpable on the day of discharge.

A short discussion on favism is given and the reasonable suggestion is made that some obscure cases of acute haemolytic anaemia of undetermined origin may have been due to favism. *B. G. Maegraith.*

GENERAL PROTOZOOLOGY.

TAKOS, M. J. **Blood Parasites of some Panamanian Birds.** *J. Parasitology.* 1947, June, v. 33, No. 3, 229–30.

WEINMAN, D. & MCALLISTER, Joan. **Prolonged Storage of Human Pathogenic Protozoa with Conservation of Virulence : Observations on the Storage of Helminths and Leptospiras.** *Amer. J. Hyg.* 1947, Jan., v. 45, No. 1, 102–21. [16 refs.]

The authors report the results of a study on the preservation of various protozoa, as well as of leptospiras and helminths, by freezing. The organisms were preserved in the blood or tissues of the host after addition of an anti-coagulant, or in cultures suspended in dilute serum or fresh culture medium, amounts up to 3 ml. in volume being used. For freezing, tubes containing the organisms were immersed in an alcohol bath brought to the desired temperature by the addition of pieces of solid carbon dioxide, after which they were stored at −70–76°C. Thawing was accomplished in a water bath at 40–42°C. The contents of the tubes were then tested for viability of the organisms by direct examination, by culture and by animal inoculations. Positive results were obtained with the following protozoa, stored for periods varying from months to several years : (*a*) trypanosomes (*T. rhodesiense, T. gambiense, T. cruzi* and *T. lewisi*), each species having been preserved in blood from infected animals and in cultures ; (*b*) leishmanias (*L. tropica* : cultures ; *L. donovani* : infected tissues) ; (*c*) *Trichomonas hominis* (cultures) ; (*d*) *Plasmodium lophurae* (infected blood). Successful results were also obtained with leptospiras : *Leptospira icterohaemorrhagiae* (cultures and infected liver) and *L. biflexa* (cultures). Larval helminths (*Wuchereria bancrofti, Dirofilaria immitis, Litomosoides carinii, Ancylostoma caninum* and *Strongyloides stercoralis*) have also been stored successfully. However, some protozoa (*Entamoeba histolytica, Toxoplasma, Balantidium coli, Trichomonas vaginalis, Chilomastix mesnili*) did not survive. It is noted that preservation by freezing enables numerous strains of parasites to be maintained with very little trouble for prolonged periods of time. *C. A. Hoare.*

CALUBAQUIB, Prudencia B. & ROLDA, H. **The Incidence of Intestinal Parasitism among Food Handlers.** *J. Philippine Med. Ass.* 1947, Apr., v. 23, No. 4, 149–52.

Between October 1945 and April 1946, the authors examined the stools of 819 Chinese and 3,862 Filipino food handlers in the Manila Public Health Laboratory. The results of their investigation are set out in two tables indicating, respectively, the incidence of intestinal parasites and of multiple infections by them found in each race.

The most prevalent parasite was *Ascaris lumbricoides* which accounted for 55·6 and 60·6 per cent. in Filipinos and Chinese respectively : the corresponding figures for *Trichuris trichiura* were 22·5 and 25·4. *Clonorchis sinensis* was found three times in Chinese and only one cestode infection was encountered—a *T. saginata* in a Filipino. Hookworms amounted to 6·2 per cent. in Filipinos and 3·2 per cent. in Chinese. The commonest double infection was *Ascaris* with *Trichuris*, which occurred in 48 per cent. of Filipinos and 74 per cent. of Chinese. Many other combinations were found.

The authors consider that the incidence of *Entamoeba histolytica* cases and carriers was " surprisingly high " at rates of 2·3 and 2·8 per cent. in Filipinos and Chinese respectively. They recommend that amoebic carriers should not be permitted to be public food handlers, and that the latter should be examined periodically. [No evidence is given to show whether vegetative or cystic forms of *E. histolytica* were encountered : the " surprisingly high " figures, which the authors attribute to insanitary conditions during the war, are not remarkable ; recent figures from Ceylon, Peru and Honduras (this *Bulletin*, 1947, v. 44, 717 ; 818) were considerably higher, and many workers

have found *E. histolytica* in the neighbourhood of 10 per cent. of stools examined in continental U.S.A.] *H. J. O'D. Burke-Gaffney.*

BIJLMER, Johannes. Over het opsporen van protozöen en eieren van enkele wormsoorten in de menselijke faeces. [**The Detection of Protozoa and Helminth Ova in Human Faeces.**] [Thesis for Doctorate of Medicine, Univ., Amsterdam, 19th June, 1947.] 98 pp., 4 figs. [Bibliography.] English summary pp. 77–80. 1947. Amsterdam: P. H. Vermeulen, De Academische Boekwinkel.

This monograph constitutes a thesis submitted by Dr. Bijlmer for the degree of M.D. in the University of Amsterdam and represents the results of his work in the Institute of Tropical Hygiene in that city. The purpose of the work was to investigate the best technique for detecting human intestinal parasites; and in seven chapters the author, having discussed the literature at length, describes the various methods employed, the results obtained and the conclusions drawn. There are numerous informative tables in the text, summaries in English, French and German, and 9 pages of references. The last chapter, which deals briefly with a small survey in Amsterdam, refers to a number of unusual findings and is illustrated with four simple figures.

In the investigation of protozoa, the author compared four concentration techniques; Bayer's, Faust's zinc-sulphate floatation, Otto's (a modification of Faust's) and a simplified Faust technique, in which washing with water was omitted and the faeces were suspended directly in 33 per cent. $ZnSO_4$ and centrifuged.

Faust's method was the best, but the simplified form gave good results: the latter modification, indeed, saved time and it is claimed that the whole process occupies only 10 minutes—no longer than a direct iodine film—compared with 25 minutes for the original Faust and for Bayer's method.

The concentration methods were not found very effective in the case of *Giardia* infections. Better results were obtained when patients were purged.

Except in the case of *Trichomonas* and *Dientamoeba fragilis*, four concentration examinations of faeces on four different days located 90 to 95 per cent. of the infections, compared with 40 to 50 per cent. with a single direct examination by eosin and iodine. Culture methods were not found practicable.

The author concludes, with regard to the finding of protozoa in faeces, that the best routine procedure is first to examine an eosin film for trophozoites; if this is negative and sufficient cysts are present, an iodine film is examined; if cysts are scanty or absent, the simplified zinc-sulphate concentration is employed, if necessary for four successive days. Purging is desirable in the case of *Trichomonas hominis* and *Dientamoeba fragilis*, which do not form cysts.

The author finds concentration methods of little advantage in the case of helminth ova, but recommends Brug's method [this *Bulletin*, 1922, v. 19, 246] for *Strongyloides* larvae, as he found the zinc-sulphate floatation unreliable for this purpose. All floatation methods failed with unfertilized *Ascaris* eggs. The author doubts whether concentration of the eggs mentioned is "sensible". "One Ascaris female lays so many eggs that they can always be spotted in a direct preparation." Regarding hookworm eggs, he writes: "it may be taken for granted that at every infection of clinical significance so many worms are present that the eggs can already be located in the direct examination." The author concludes that for *Ascaris* and hookworms, an ordinary water preparation suffices.

Finally a note is given regarding a survey of several hundred stools among the population of Amsterdam. In one series, 76 per cent. of specimens showed intestinal protozoa, of which *E. histolytica* constituted 5 to 10 per cent.

The investigation dealt with 878 samples of stools from 478 persons. In 378 cases (78 of these patients were from Japanese camps), a single examination was made ; in the remaining 100, five examinations were made in each case.

The monograph contains a large amount of useful practical information.

H. J. O'D. Burke-Gaffney.

JOHNSON, J. G. **The Physiology of Bacteria-Free** *Trichomonas vaginalis.* *J. Parasitology.* 1947, June, v. 33, No. 3, 189–98, 4 figs. [10 refs.]

HOGUE, Mary J. **The Behavior of** *Trichomonas vaginalis* **in Tissue Cultures : a Movie.** *J. Parasitology.* 1947, June, v. 33, No. 3, 199–200.

GENERAL ENTOMOLOGY.

FRIZZI, G. **Salivary Gland Chromosomes of** *Anopheles.* [Correspondence.] *Nature.* 1947, Aug. 16, 226–7, 1 fig.

WEBB, J. E. **A Spraying Apparatus and Testing Chamber for investigating the Residual Action of Insecticidal Deposits.** *Bull. Entom. Res.* 1947, Aug., v. 38, Pt. 2, 209–32, 3 figs. on 1 pl. & 9 text figs.

The apparatus makes use of an " Aerostyle " pneumatic spray gun to atomize insecticide into a vertical tower. The dimensions of the tower permit objects up to 20 cm. square to be treated, and these can subsequently be built up into a cage if necessary to use with flying insects. Applications are made by droplets settling out of the mist produced by the atomizer. To prevent undue evaporation of the droplets, the tower is pre-sprayed to saturate the air in it. Procedure therefore is : (1) Pre-spray (5 charges of water for aqueous sprays, or 1 charge of kerosene for kerosene sprays) ; (2) wait ten minutes ; (3) insert object for treatment on a sliding tray ; (4) spray suitable charge ; (5) allow spray to settle for 5 minutes, then withdraw tray ; (6) wait 5 more minutes then insert *next* article for treatment . . . and so on. This allows a routine spray treatment every ten minutes.

The apparatus is calibrated for variations of deposit due to various factors : spraying pressure, temperature, time of exposure to mist, volume of liquid sprayed, etc., and appears to be reliable in performance. The subject is discussed in great detail, which should be sought in the original.

J. R. Busvine.

BOCAT, R. Étude sur le DDT (4-4′ *Dichlodiphenyltrichlorethane*), découverte-propriétés-dosage-toxicité-emploi. [**The Discovery, Properties, Dosage, Toxicity and Method of Employment of DDT.**] *Méd. Trop.* Marseilles. 1947, May–June, v. 7, No. 3, 263–70.

A general account.

LOVETT, W. C. D. **Control of Mosquito Breeding by the Use of D.D.T. Solution absorbed on Briquettes.** *East African Med. J.* 1947, May, v. 24, No. 5, 196–8.

In a wide area of British Somaliland a great deal of mosquito breeding occurs in wells, and a control method was desired which should be simple enough to delegate to local police or village elders. The method described has the advantage that the kerosene used with the DDT cannot be diverted to illegitimate uses.

Small briquettes are prepared by mixing sawdust and plaster of paris (2 : 1) before wetting ; the mixture is allowed to set in a shallow layer and is cut up into one-inch cubes. These cubes are soaked in a solution of 5 per cent. DDT in diesoline ; it was found that they absorbed about 7½ cc. per cube, which was enough to treat four square yards of water surface. Treatment of wells is simply done by dropping in one or more cubes each month, according to size. Preliminary results were very satisfactory. In the table of results, only culicine mosquitoes and larvae are mentioned. *J. R. Busvine.*

HACKMAN, R. H. **The Persistence of DDT on Cattle.** Reprinted from *J. Council Scient. & Indust. Res.* 1947, Feb., v. 20, No. 1, 56–65, 2 figs. [13 refs.]

"Evidence is brought forward to show that the most important factor causing the removal of DDT from cattle sprayed with DDT preparations is licking, either by the animal concerned, or by another animal. Other factors have been investigated, viz., solar radiation and rain, growth and loss of hair, rubbing, production of skin secretions, absorption into the skin and hair and flaking of the epithelium, but these have been shown to play a minor rôle. It is concluded that the amount of DDT ingested by licking would not be sufficient to produce toxic symptoms."

EDNEY, E. B. **Laboratory Studies on the Bionomics of the Rat Fleas,** *Xenopsylla brasiliensis*, **Baker, and** *X. cheopis*, **Roths. II. Water Relations during the Cocoon Period.** *Bull. Entom. Res.* 1947, Aug., v. 38, Pt. 2, 263–80, 2 figs. [25 refs.]

In a previous paper [this *Bulletin*, 1945, v. 42, 504], the author in his summary stated that "Saturation deficiency during the pre-pupal stage is shown to bear a linear relationship to adult longevity," and the suggestion was put forward that during the period between cocoon formation and pupation the flea larvae lose water in relation to the saturation deficit (s.d.) of the air, the water reserve of the emerging adult depending on the s.d. during this short pre-pupation period. The present paper is an account of experiments undertaken to confirm or disprove this suggestion and to obtain more precise data regarding the water relations during the cocoon period. The ingenious methods by which these data were obtained and studied are fully described, but should be sought in the original since they are not easily summarized. These new experiments have enabled the author to confirm his previous findings that in unfed adult *Xenopsylla*, longevity varies according to the humidity experienced during the cocoon period. On the other hand, these experiments yield proof that the cause lies in the loss of water during the pupal and not, as previously suggested, during the pre-pupal period. On the contrary, within 24 hours of cocoon formation the prepupa begins to absorb water vapour from the air, and continues to do so until the prepupa moults to become a true pupa. This process of water absorption, however, can only occur at a relative humidity of above about 45° per cent. at 12°, 24°, and 35°C. Above this figure the prepupae are incapable of absorbing water and become desiccated. It is significant that 45 per cent. R.H. is the critical one at all temperatures tested, since it shows that the process of absorption of water is not limited by one saturation deficit.

MELLANBY [this *Bulletin*, 1932, v. 29, 823], has shown that, whereas adult *Xenopsylla* can withstand exposure to 38°C. for 24 hours, at all humidities, the larva can only withstand 22°C. in dry air, with a regular rise in their thermal

death point to 36°C. at 90 per cent. R.H. BUXTON [this *Bulletin*, 1939, v. 36, 308], confirmed these findings and showed that low humidities adversely affect the larvae. Edney points out that these observations suggest that the feeding larvae are water-losers, but that the change to the water-gaining prepupae may occur slowly during or towards the end of the last larval instar; and that this is a subject which deserves further study.

The latter part of Dr. Edney's paper deals with the result of experiments designed to show the water relationships after pupation. From these results it appears that fleas lose water after pupation, for the percentage water content decreases with the total weight lost. This rate of water loss shows a great deal of variation, and considerable evidence is adduced to show that more is lost in dry than in moist air. Thus, adults emerging from pupae kept at higher humidities weighed more, had higher percentage water contents, and lived longer without food, than was the case amongst fleas emerging from pupae kept at lower humidities. *R. M. Gordon.*

BOOK REVIEWS.

NAPIER, L. Everard [C.I.E., F.R.C.P., etc.] & DAS GUPTA, C. R. [M.B., D.T.M. (Calcutta), etc.]. **Haematological Technique.** 3rd Edition, Revised by C. R. DAS GUPTA, M.B., D.T.M. (Calcutta). pp. viii+128+ix–x+2, 38 figs. & 3 coloured pls. 1945. Calcutta: U.N. Dhur & Sons Ltd., 15, Bankim Chatterjee Street. [*Rs.* 8/-.]

This book deals with the routine haematological methods employed at the Calcutta School of Tropical Medicine; the original edition was formed by the collection of a series of haematological studies published during 1940 and 1941 in the *Indian Medical Gazette.*

The first chapter deals with the collection of blood, emphasis being placed on the advantages of using venous samples. The following chapter records various methods of determining haemoglobin and a praiseworthy plea is made for recording the results in grammes rather than in percentages. The use of the Hellige instrument is fully described, as this is the one used in the authors' laboratory; but although several other instruments and various methods are mentioned, little detailed discussion of their relative merits is given. Chapters then follow on methods for counting the various cells of the blood, for determining erythrocyte volume and for ascertaining the bilirubin content of the blood. In these chapters not only are the actual procedures clearly and carefully described, but also valuable information is given on the care and management of the instruments employed and on the preparation of the various solutions used in the examinations. Following this there are chapters on the making and staining of blood films, and on the methods of performing differential white cell counts. The development of the various blood cells and their identification both in smears of the peripheral blood and of the bone marrow are then described. A chapter also deals ably with the technique of sternal puncture. At this point, one may remark on the extraordinary mixture of ignorance and intelligence that the authors appear to assume on the part of their readers. On the one hand, simple procedures, for example, how to note down figures on a piece of paper during a cell count, are described in extreme detail; while, on the other, difficult matters requiring much skill and experience for their accurate and trustworthy performance, such as the identification and differentiation of the various types of developing blood cells, are also dealt with.

The value and wisdom of the authors' assumption doubtlessly rests upon their long and wide experience of teaching, and should give their book a special place, particularly in schools, where western medicine is a comparatively recent introduction, for the *ab initio* training of students in haematology.

Further chapters in the book deal with miscellaneous matters, such as gastric analysis in relation to anaemia, erythrocyte sedimentation rate, erythrocyte fragility tests, bleeding and coagulation times, erythrocyte diameters, blood grouping and transfusions. The final chapter gives a scheme for the haematological investigation of a patient. A short bibliography is also appended. There is no index, in the usual sense, to contents of the book, but this is no serious disadvantage since, in addition to the chapter headings, there are subheadings which make reference to any particular subject quite easy. Since the book was designed for use in India, certain haematological procedures, for example methods of examining for sicklaemia, are omitted. In the text there are, unfortunately, a number of typographical errors and the photographic reproductions and coloured diagrams are of poor quality, the colours in the latter appearing out of register. This may be explained on the grounds that the present, and in fact each of the previous editions, were produced during the war. *F. Murgatroyd.*

WESENBERG-LUND, C. [Prof. an der Universität Kopenhagen]. **Biologie der Süsswasserinsekten.** [Biology of Freshwater Insects.] 682 pp., 13 pls. & 501 figs. 1943. Copenhagen: Gyldendalske Boghandel. Nordisk Forlag. Berlin: Vienna: Verlag J. Springer. [36*s*.]

More than 30 years ago the author produced a volume in Danish dealing with insect life in fresh water. All those who could read it found this book a most valuable standby and will recognize that though the present work has been very largely re-written it is in form and spirit the direct descendant, indeed almost the second edition, of the earlier book.

The greater part of the present book, running to nearly 600 pages, takes all those Orders or Families of insects which include any aquatic members, and describes the life-history and also the anatomy in so far as it is closely related to biology. The book contains a wealth of information on those groups which are abundant or varied in fresh water: for instance, there are forty pages on Chironomidae, containing most valuable information on those characters in the early stages, which separate the sub-families. On the mosquitoes there are 65 pages. Other groups which are not generally aquatic, but contain a few aquatic members, are dealt with more briefly.

Though the book is founded very largely on the author's long experience in Denmark, it by no means neglects interesting work from all parts of the world. One observes, for instance, an account of those extraordinary grasshoppers found in several parts of the world, but not in Europe, some of which skim the surface of water and others which are actually sub-aquatic.

The book is concluded by chapters on topics of general interest, such as the characteristics of the insects of lakes, running waters and the water surface: it also deals with respiration, hibernation and similar general problems.

The book is the work of a man who has worked for a very long period at his subject and generally in the open air. The point of view is that of a naturalist and there is not much reference to modern physico-chemical work or experimental ideas, nor to the applied side of the subject. There are a number of valuable figures, particularly those which compare the larvae of aquatic insects. The references given at the end of the book are carefully selected: they include a proportion of recent papers. *P. A. Buxton.*

WISELOGLE, Frederick Y. [Edited by]. **A Survey of Antimalarial Drugs 1941-1945.** Subsidized by the Office of Scientific Research and Development on Recommendation by the Committee on Medical Research. Vol. I. pp. xi+536. Vol. II. Part 1. pp. 1–987. Part 2. pp. 988–1921. 1946. Ann Arbor, Michigan: J. W. Edwards. [$30.00.]

This publication contains an enormous amount of information, the scope of which is indicated by the following extract from the editor's preface :—

"A Survey of Antimalarial Drugs, 1941–1945 presents in organized form the scientific achievements of a vast, co-ordinated, wartime research program. . . .

"The monograph was prepared with two main objectives constantly in mind: (1) to expedite the search for still better antimalarial drugs and (2) to serve as an established pattern for systematic chemotherapeutic studies of other infections.

"The monograph contains all data in the files of the Survey of Antimalarial Drugs which were released for publication by the co-operating research groups. No information of any consequence has been withheld. Material which was obtained as a part of the co-ordinated program but which does not lend itself to systematic presentation, such as methods of synthesis of compounds, biochemical and parasitological studies, and intensive pharmacological and clinical studies on certain compounds, will be published separately by the individual authors. The identification of substances by their Survey Numbers should facilitate the location of a substance of interest in both the monograph and current literature."

The first volume contains a history of the co-operative wartime programme, an account of pharmacological investigations, a discussion of chemical series of potential interest, and of the rationale underlying the clinical evaluation of antimalarial drugs, a summary (of more than 230 pages) of data on the drugs tested in man, and descriptions of the various testing procedures. Volume 2 (in two parts) contains tables, and very full indexes (comprising over 300 pages).

The Survey should be in the library of every organization engaged in chemotherapeutic research. *Charles Wilcocks.*

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Bart.	,,	Bartonellosis.
Bl.	,,	Blackwater.
B.R.	,,	Book Review.
Chl.	,,	Cholera.
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Der.	,,	Dermatology and Fungous Diseases.
Dys.	,,	Dysentery (Bacillary and Unclassed).
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Arnold, E. H., with Ferguson & Upholt, 645 (Mal.)
——, Simmons, S. W. & Fawcett, D. G., 277 (Mal.)
Arnold, W. T. & Levy, M. D., 237 (Der.)
Astrup, A., 836 (Hel.)
Attiah, M. A. H., 614 (Oph.)
——, Salem, H. H. & El-Gammal, Y., 618 (Oph.)
Aubin, H., with Raynaud & Pegullo, (718) (R.F.)
Audibert, Y., with Sautet, 131 (Ent.)
Audy, J. R., 577 (Typh.)
Augustine, D. L., with Lowe, (754) (Misc. Dis.)
Austin, C. J., 728 (Lep.)
Australia, Commonwealth of, 476 (B.R.)
Autheman, with Quérangal des Essarts & Soubigou, (664) (Am.)
——, with Soubigou & Guennec, 585 (Am.)
Avery, J. L., 445 (Hel.)
Ayadi, M. S. & El Kordy, M., (312) (Am.)
de Azevedo, J. F., 981 *bis*, 984 (Leish.)
—— & Teixeira, A. W. G., 981 (Leish.)
Aziz, M. A., 465 (Ent.)

B.

Bachi, R., 542 (Ent.)
Bacigalupo, J., Rey, M. & Toce, A., 529 (Hel.)
Badinez, S., O., (511) (Tryp.)
Bado, J. L., 223 (Hel.)
Baer, J. E., with Brodie & Udenfriend, 793 (Mal.)
Baert, H., with Wittebolle, 821 (R.F.)
Baez, M. M., with Hinman, 371 (Reports, etc.)
Bahmanyar, M., with Baltazard & Mofidi, 905 (R.F.)
——, with ——, —— & Seydian, 906 (R.F.)
Bahrami, A., 1021 (Lab.)
Bailey, K. P., 657 (Y.F.)
Bailly, J., with Remlinger, 949, 950, 1030 (Rab.)
Baker, E. E., Sommer, H., Foster, L. E., Meyer, E. & Meyer, K. F., 712 (Pl.)
Baker, J. A., 896 (Typh.)
Baker, M., with Brackett & Waletzky, 291 (Mal.)
——, with ——, ——, Hughes & Williams, 40 (Mal.)
Balamuth, W., 1066 (Am.)
——, & Howard, B., 582 (Am.)
Balcet, C., (618) (Oph.)
Ball, E. G., 400 (Mal.)
——, with Anfinsen, Geiman, McKee & Ormsbee, 1043 (Mal.)
——, with Geiman, Anfinsen, McKee & Ormsbee, 1043 (Mal.)
——, with McKee, Ormsbee, Anfinsen & Geiman, 1043 (Mal.)
Ball, G. H., 1045 (Mal.)
Ballantine, I. D., with Coghill & Lawrence, 718 (R.F.)
Ballif, L., with Ciuca & Chelarescu, 561 (Mal.)
——, with ——, —— & Cristescu, 559 (Mal.)
Bals, M., with Potop, 301 (Typh.)
Baltazard, M., 92, 1005 (R.F.)
——, Mofidi, C. & Bahmanyar, M., 905 (R.F.)
——, ——, —— & Seydian, B., 906 (R. F.)
Balthazar, E., with Payne & Bezerra, 502 (Mal.)
——, with —— & Fernandes, 502 (Mal.)
Bami, H. L., Iyer, B. H. & Guha, P. C. (793) (Mal.)
Banerjee, A., with Chatterjee, (989) (Typh.)
Banerjee, S. & Ghosh, N. C., 452 (Def. Dis.)
Bang, F. B., with Ferguson, Graham & Hairston, 435 (Hel.)
—— & Hairston, N. G., 435 (Hel.), 1038 (Mal.)
——, ——, Graham, O. H. & Ferguson, M. S., 435 (Hel.)
——, ——, Maier, J. & Trager, W., 281, 503 (Mal.)
——, ——, Trager, W. & Maier, J., 696 (Mal.)
——, with Trager & Hairston, 1036 (Mal.)

Bannerman, H. C., 547 (Reports, etc.)
Baranger, P., (1036) (Mal.)
Barber, F., 736 (Hel.)
Barber, H. J. & Wragg, W. R., (35) (Mal.)
Bardenat, C., with Manceaux & Susini, 849 (Misc. Dis.)
Barlovatz, A., 810 (Typh.)
Barlow, C. H. & Abdel Azim, M., 831 (Hel.)
——, with —— 832 (Hel.)
Barlow, F. & Hadaway, A. B., 1053 (Tryp.)
——, with ——, 1038 (Mal.)
Barman, J. M., 908 (Lep.)
Barnes, G. T., 841 (Def. Dis.)
Barnett, G. S., with Morgan & Sheppard, 1039 (Mal.)
Barnhart, J. M., (1030) (Rab.)
Barnhill, K. G., with Elmendorf & Takos, 882 (Mal.)
Barondes, R. de R., (726) (Lep.)
Baroody, B. J., 470 (Lab.)
—— & Most, H., 333 (Hel.)
Barretto, M. P., with Pessôa, 300 (Leish.)
Barthel, W. F., with Gersdorff, 467 (Ent.)
Bartter, F. C., Cowie, D. B., Most, H., Ness, A. T. & Forbush, S., 1012 (Hel.)
Basnuevo, J. G. & Sotolongo, F., 585, (663) (Am.)
Bastos da Luz, J. V. & Nogueira, J. F. P. (934) (Der.)
——, with Salazar Leite, 932, (934) (Der.)
Basu, B. C., 1030 (Mal.)
Basu, N. K. & Lal, S. B., (860) (Reports, etc.)
—— & Sharma, S. N., (860) *bis* (Reports, etc.)
Basu, P. N., with Sen & Chakraborty, 209 (Chl.)
Bates, M. & Roca-Garcia, M., 308, 310, 415 (Y.F.)
——, with ——, 580 (Y.F.)
Batista, D., 875 (Mal.)
Baud, C., with Coudert, 241 (Ent.)
——, with ——, Bozicevich & Brady, 833 (Hel.)
——, with ——, McMullen & Faust, 832 (Hel.)
Bayliss, M., with Nelson, Webb & Starkey, 229 (Hel.)
Baz, I. I., (337) (Hel.)
——, with Halawani & Morcos, 792 (Mal.)
Baz, I., with Halawani & Morkos, 698 (Mal.)
Bazemore, J. M., Johnson, H. H., Swanson, E. R. & Hayman, J. M., Jr., 559 (Mal.)
Bean, W. B., with Kark, Aiton, Henderson, Johnson & Richardson, 842 (Def. Dis.)
Beard, R. R., with Smith, Rosenberger & Whiting, 537 (Der.)
——, with ——, Whiting & Rosenberger, 675 (Der.)
Beato Gonzalez, F., 622 (Misc. Dis.)
Beaudiment, R., Laviron, P. & Aretas, R., 727 (Lep.)
Beck, M. D. & van Allen, A., 893 (Typh.)
Beck, O. H., with Leavitt, 916 (Hel.)
Becker, E. R., Burks, C. S. & Kaleita, E., 177 (Mal.) 583 (Am.)
Becker, F. T., with Glenn, Kaplan & Read, 167 (Mal.)
——, with Kaplan, Read & Boyd, 25, 281 (Mal.)
—— —— & ——, with ——, 280 (Mal.)
Becker, F. T., with Read & Kaplan, 177 (Mal.)
Bedford, D. E., Aidaros, S. M. & Girgis, B., 433 (Hel.)
Beemer, A. M., 1068 (Am.)
Beet, E. A., 1091 (Haem.)
Begg, A. M., with van den Ende, Stuart-Harris, Fulton, Niveh, Andrewes, Elford, White, Hawley, Mills, Hamilton & Thomas, 472 (B.R.)
Beklemischev, V. N., 639 (Mal.)
Bell, E. J., Bennett, B. L. & Whitman, L., 514 (Typh.)
Bell, J. A., with Olson & Emmons, 848 (Der.)
Bellerive, A. & Dambreville, C., 16 (Mal.)
Beltrán, E. & Dávalos, A., 960 (Mal.)
——, Dávalos, A. & Sandoval, C. A., 968 (Mal.)
—— & Sandoval, A. M., 160, 961 (Mal.)
Bénard, H. Rambert, P., Cajdos, A, & Van den Broucke, 533 (Haem.)
Bénard, R. & Kerbrat, G., (514) (Typh.)
Bendich, A. & Chargaff, E., 407 (Typh.)
Benedek, T., with Lewe, I., Price, H., Koransky, A. & Van Horn, F., 458 (Der.)
Bengtson, I. A., 60, 61, 302, 304, 812 (Typh.)
Benhamou, E., Destaign, F., Leonardon, H. & Vargues, R., (586) (R.F.)
——, Nouchy & Sebah, P., 238 (Misc. Dis.)
Benitez Calvo, L. A., with Piedrola Gil, (636) (Rab.)
Benmussa, S., with Durand & Caruana, 56 (Leish.)
Bennet, E. A., 360 (Misc. Dis.)
Bennet, J., 360 (Misc. Dis.)
Bennett, B. L., with Bell & Whitman, 514 (Typh.)
——, with Plotz, Smadel, Reagan & Snyder, 204 (Typh.)
——, with Smadel, Jackson & Rights, 513 (Typh.)
Bequaert, J. C., 682 (Ent.)
——, with Veatch & Weinman, 293 (Tryp.)
Bercovitz, Z. T. & Schwachman, H., 338 (Hel.)
Bereston, E. S. & Cheney, G., 348 (Der.)
—— & Waring, W. S., (350) (Der.)
Bergeret, C. & Raoult, A., (426) (R.F.)
Berke, Z., 512 (Typh.)
Berkowitz, A. P., 513 (Typh.)
Berlin, C., (675) (Der.)
Bermudez, O., with Andreon, (836) (Hel.)
Bernal, M., with Mom, 596 (Lep.)
Bernkopf, H., Olitzki, L. & Stuczynski, L. A., 587 (R.F.)
Bernstein, J., Pribyl, E. J., Losee, K. & Lott, W. A., (964) (Mal.)
——, Stearns, B., Dexter, M. & Lott, W. A., (964) (Mal.)
——, ——, Shaw, E. & Lott, W. A., (964) (Mal.)
Bertram, D. S., with Williamson & Lourie, 970 (Mal.)
Bertrand, (1080) (Hel.)
Besdenjesznich, K. V., with Fajerstein, 409 (Typh.)
Bethell, F. H., Meyers, M. C., Andrews, G. A., Swendseid, M. E., Bird, O. D. & Brown, R. A., (533) (Haem.)
——, with Swendseid, Bird & Brown (533) (Haem.)

Bezerra, D. A., with Payne & Balthazar, 502 (Mal.)
Bhaduri, N. V., with Mukerji, 836 (Hel.)
——, with —— & Narain, 599 (Hel.)
Bhaduri, P. N., with Greval, 239 (Misc. Dis.)
Bhatt, B. L. & Nargund, K. S., (666) (Hel.)
Bhattacharji, L. M., Singh, J. & Sen Gupta, G. P., 546 (Lab.)
Bhide, B. V., with Pendse, Gokahle & Phalnikar, (681) (Ent.)
Bhore, J., 252 (B.R.)
Bianco, A. A., with Saunders & Jordan, 108 (Hel.)
——, ——, Levine, A. S. & Cohn, R., 1034 (Mal.)
Bichovsky, L., with Adler & Sadovsky, (624) (Prot.)
Bichowsky, L., with Adler, 52 (Tryp.)
Biggam, A., 249 (Reports, etc.), 360 (Misc. Dis.)
Bignall, J. R., with Fleming & Blades, 711 (Den.)
Bijlmer, J., 111 (Hel.), 1101 (Prot.)
Billi, A., (168) (Mal.)
Billings, T., with Winkenwerder, Hunninen, Harrison, Carroll & Maier, 834 (Hel.)
Bimbad, S. M., 214 (Lep.)
Binet, L. & Wellers, G., (122) (Vms.)
Bird, O. D., with Bethell, Meyers, Andrews, Swendseid & Brown, (533) (Haem.)
——, with Swendseid, Brown & Bethell (533) (Haem.)
Birkett, B., with Bishop, 969 (Mal.)
Birnbaum, D., Goldblum, N. & Kligler, I. J., 22 (Mal.)
——, with Mer & Aioub, 542 (Ent.)
Birnkrant, W. B., with Shookhoff & Greenberg, 603 (Hel.)
Birstein, O., with Elderfield, Kremer, Kupchan & Cortes, (964) (Mal.)
Bishop, A. & Birkett, B., 969 (Mal.)
Bishopp, F. C., (369) (Ent.)
Bissell, H. D., with Carlson & Mueller, 39, 174 (Mal.)
Black, D. A. K., 233 (Sp.)
——, Bound, J. P. & Fourman, L. P. R., 1085 (Sp.)
——, with McCance, 44 (Bl.)
Black, R. H., 18, 176 (Mal.)
Black, T. C., 336 (Hel.)
Black, W. C., Florio, L. & Stewart, M. O., 899 (Den.)
Blackman, G. G. B., with Stevens, 169 (Mal.)
Blades. A. N., with Fleming & Bignall, 711 (Den.)
Blair, D. M., with Alves, 826, 827 (Hel.)
Blair, M., with Hauschka & Saxe, 1054 (Tryp.)
Blanc, F. & Siguier, F., (522), 524 (Am.)
Blanc, G., (758) (Reports, etc.)
—— & Delage, B., 931 (Vms.)
—— & Martin, L. A. (813) (Typh.)
——, —— & Maurice, A., 709, 898 (Typh.)
Blank, H., 935 (Ulc.)
Blaschko, H., Chou, T. C. & Wajda, I., (698) (Mal.)
Blaxter, K. L., 754 (Ent.)
Block, S. S., 467 *bis* (Ent.)
Bloss, J. F. E., 527 (Ys.)
Blumberg, R. W., with Sabin, 814 (Den.)
Board of Trade, 624 *ter* (Ent.)
Boase, A. J. (849) (Der.)
Bobliolo, L., 349 (Der.)
Bocat, R., (1102) (Ent.)
Boechat, W. M., 218 (Hel.)
Boeing, P. J., with Sashin, Brown, Laffer & McDowell, 460 (Der.)
de Boer, C. J., Kunz, L. J., Koprowski, H. & Cox, H. R., 712 (Den.)
Boiron, H., 429, 586 (R.F.)
——, with Durieux & Koerber, 991 (Y.F.)
Bolivia, 1024 (Reports, etc.)
Bol. Oficina Sanitaria Panamericana, 301, 703 (Typh.)
Bomford, R. R., 360 (Misc. Dis.)
Bond, V. P., Bostick, W., Hansen, E. L. & Anderson, H. H., 585 (Am.)
Bone, G., with Rodhain, (807) (Tryp.)
Bonne, C., 584 (Am.), 932 (Der.)
——, Bras, G. & Lie Kian Joe, 1013 (Hel.)
Bonnet, D. D. & Worcester, D. J., 366 (Ent.)
Bonnin, H., Dubourg, E. & Moretti, G. F., 343 (Haem.)
Bonnin, J. M., with Andrew & Williams, 202 (Typh.)
von Bonsdorff, B., 1081 (Hel.)
Bonsmann, M. R., (458 *bis*) (Vms.)
Boquet, P., Girard, O. & Corvazier, R., 235 (Vms.)
de Borchgrave, O., 570 (Tryp.)
Borell, A. E. & Bryant, M. D., (755) (Ent.)
von Bormann, F. & Preuss, H., 891 (Typh.)
Bose, B. C., Iyengar, N. K. & Mukerji, B., 189 (Leish.)
Bose, R., with Dharmendra & Sen Gupta, 189 (Leish.)
Bossak, H. N., with Rein, 136 (Lab.)
Bostick, W., with Bond, Hansen & Anderson, 585 (Am.)
Bound, J. P., with Black & Fourman, 1085 (Sp.)
Bounds, L. H. & Kingery, R. M., (429) (R. F.)
Bourgain, M., 212, 587 (R. F.)
Bourova, L. F., 313 (Am.)
Bovarnick, M. R., with Hellerman & Porter, 400 (Mal.)
Bowden, K. M., 168 (Mal.)
Bowles, R. V., with Dick, 643 (Mal.)
Boyd, M. F., with Becker, Kaplan & Read 280 (Mal.)
——, with Kaplan, Read & Becker, 25, 281 (Mal.)
—— & Kitchen, S. F., 560 (Mal.)
Bozicevich, J., Donovan, A., Mazzotti, L., Diaz, A. F. & Padilla, E., 669 (Hel.)
——, with Wright, Brady & Bauman, 833 (Hel.)
Bracey, P., with David, 133, 544 (Ent.)
Brachetto-Brian, D. & Schena, A. T., 108 (Hel.)
Brackett, S., with Hughes, 41 (Mal.)
——, Waletzky, E. & Baker, M., 291 (Mal.)
—— & ——, with ——, Hughes, C. O. & Williams, H., 40 (Mal.)
Bradley, G. H. & Fritz, R. F., 180 (Mal.)
Brady, F. J., with Jones, 217 (Hel.)
——, with Wright, Bozicevich & Bauman 833 (Hel.)

Braithwaite, P., 961 (Mal.)
von Brand, T., Rees, C. W., Reardon, L. V. & Simpson, W. F., 84 (Am.)
—— & Simpson, W. F., (669) (Hel.)
Brandt, J. L., 667 (Hel.)
Bransfield, J. W., with Dorrance, 602 (Hel.)
Bras, G., with Bonne & Lie Kian Joe, 1013 (Hel.)
Braun-Blanquet, M., with Harant & Giroux, 1013 (Hel.)
Bray, W. E., Jr., with Mider & Smith, 610 (Der.)
Brazzaville [A. E. F.], 45 (Tryp.) 75 (Y.F.)
Brelet, M., 1004 (Am.)
Brennan, D. J., 115 (Def. Dis.)
Brescia, F., 881 (Mal.)
Briceño-Iragorry, L., with Briceño Rossi, (702) (Typh.)
Briceño Rossi, A. L. & Briceño-Iragorry, L., (702) (Typh.)
Brindle, H. & Rigby, G., 370 (Lab.)
Brisou, J., 222 (Hel.)
Bristowe, W. S., 364 (Ent.)
British Guiana, 181, 504 (Mal.), 658, 994 (Y. F.)
British Med. J., 616 (Oph.), 719, 824 (Lep.), 741 (Sp.)
Brochen, L., with Robin, 643 (Mal.)
Brockman, J. A., Jr., with Keopfli & Mead, (964) (Mal.)
Brodie, B. B., Udenfriend, S. & Baer, J. E., 793 (Mal.)
——, —— & Dill, W., 793 (Mal.)
——, ——, —— & Chenkin, T., 793 (Mal.)
——, ——, —— & Downing, G., 793 (Mal.)
——, with —— & Josephson, 793 (Mal.)
——, —— & Taggart, J. V., 793 (Mal.)
Brooke, M. M. & Proske, H. O., 22 (Mal.)
——, with Richardson, Hewitt, Seager, Martin & Maddux, 289 (Mal.)
Brooks, A. G., with Ahuja, Veeraraghavan & Menon, (846) (Vms.)
Broun, G. O., with Sullivan, 532 (Haem.)
Brouwer, D., 129 (Misc. Dis.)
Brown, A. F., 314 (Am.)
Brown, A. H., with Molnar, Towbin, Gosselin & Adolph, 461 (Heat Str.)
Brown, G. N., with Sashin, Laffer, McDowell & Boeing, 460 (Der.)
Brown, H. W. & Thetford, N. D., 338 (Hel.)
Brown, M. & Rennie, J. L., 395, 497 (Mal.)
Brown, R. A., with Bethell, Meyers, Andrews, Swendseid & Bird, (533) (Haem.)
——, with Swendseid, Bird & Bethell, (533) (Haem.)
Brown, R. G., with Helwig, (442) (Hel.)
Brumpt, E. & Dao Van Ty, 184 (Mal.)
Brumpt, L., with Jude, 811 (Typh.)
Brumpt, L. C., (704) (Typh.)
Brundrett, J. C., with Maegraith, Rigby & Sladden, 886 (Leish.)
Bruneau, (655) (Typh.)
Bruneau, J., 660 (Chl.)
—— & Nguyen-Dinh-Diep, 655 (Typh.)
Brunsting, H. A., with Engstrom, Gordon & Marble, 961 (Mal.)
de Bruyn, P. P. H., (901) (Am.)
Bruynoghe, G. & Ronse, M., 822 (R. F.)
Bryant, M. D., with Borell, (755) (Ent.)
Buchanan, G., 93 (R.F.)
Buck, M., with Schnitzer & Lafferty, 185 (Tryp.)
Buckley, J. J. C., 909 (Hel.)
Budeir, R., with Haddad & Sheiban, 320 (R.F.)
Buecherl, W., 931 (Vms.)
Buechler, E., with Olitzki, 196 (Typh.)
Bueding, E., Peters, L. & Waite, J. F., 667 (Hel.)
——, with Welch, Peters, Valk & Higashi, 1015 (Hel.)
Bugher, J. C., with Haddow, Smithburn & Mahaffy, 992 (Y.F.)
Buguianishvili, S., with Kamalov, Gordadze, Tsutsunava, Khizanishvili & Kamalova, 224 (Hel.)
Bühler, F., 1061 (Typh.)
Bull. Méd. de l'Afrique Occidentale Française, 352 (Ulc.)
Bull. Office Internat. d'Hyg. Publique, 192 (Typh.)
Bull. U.S. Army Med. Dept., 89 (Am.)
Bulycheva, N. A., with Zerchaninov, 216 (Hel.)
Bumstead, J. H., with Liebow, MacLean & Welt, 351 (Ulc.)
de Burca, B., 157, 953 (Mal.)
Burch, G. E., 126 *bis* (Heat Str.)
Burdon, K. L., 721 (Lep.)
Burgess, R. C., 114 (Def. Dis.)
Burgess, R. W., 787 (Mal.)
—— & Young, M. D., (161) (Mal.)
——, with ——, 694 (Mal.)
——, with ——, Stubbs, Ellis & Eyles, 275 (Mal.)
Burgoon, D. F., with Machella & Fine, 557 (Mal.)
Burkitt, R. T., 752 (Misc. Dis.)
Burks, C., with Becker & Kaleita, 583 (Am.)
Burks, C. S., with Becker & Kaleita, 177 (Mal.)
Burks, J. W., Jr. & Thompson, P. E., 124 (Der.)
Burrows, R. B., Morehouse, W. G. & Freed, J. E., 926 (Hel.)
Burrows, W., Mather, A. N., Elliott, M. E. & Wagner, S. M., 422 (Chl.)
——, ——, McGann, V. G. & Wagner, S. M., 422 (Chl.)
Burruss, H. W. & Hargett, M. V., 994 (Y.F.)
Burtt, E., 803 (Tryp.)
——, with Fairbairn, 506 (Tryp.)
Bussell, L. J., 638 (Rab.)
Bustamante, M. E. & Varela, G., 656 (Typh.)
Butler, F. A. & Sapero, J. J., 1034 (Mal.)
Butler, M. G., 792 (Mal.)
Butler, R., Davey, D. G. & Spinks, A., 1037 (Mal.)
Buxton, P. A., 795 (Mal.), 860 (B.R.)
Byrne, E. A. J., 537 (Der.)

C.

Caballero y C., E., 230 (Hel.)
Cabannes, with Sarrouy & Roche, 807 (Leish.)
Cabasso, V., 809 *bis* (Typh.)
Cain, L. J., with Smith & Gallup, 177 (Mal.)
de Caires, P. F., 658, 994 (Y.F.)

Cairo, 831, 832 (Hel.)
Cajdos, A., with Bénard, Rambert & Van den Broucke, 533 (Haem.)
Cakrtova, M., with Wolfson, 566 (Mal.)
Calero, C., 525 (R.F.)
Calero, M. C., (932) (Der.)
California Mosquito Control Association, 758 (Reports, etc.)
Callender, S. T., 743 (Haem.)
Calubaquib, P. B. & Rolda, H., 1100 (Prot.)
Cambier, R., with Vincke & Lips, 286 (Mal.)
Cambournac, F. J. C., with Fonseca, Pinto, Pereira & Cunha, 487 (Mal.)
——, Simões, J. M. P. & Queiroz, E. J. S., 181 (Mal.)
Cambre, P. J., with Fite & Turner, 1008 (Lep.)
Cameron, D. A., with Elsdon-Dew & Horner, 310 (Am.)
Cameron, J. D. S., (684) (Reports, etc.), (714) (Am.)
Campbell, A. G., with Humphreys, 758 (Reports, etc.)
Campbell, B. K., with Campbell, K. N., Kerwin & LaForge, (34) (Mal.)
——, with ——, Sommers & Kerwin, (34) (Mal.)
Campbell, K. N., Helbing, C. H. & Kerwin, J. F., (34) (Mal.)
—— & Kerwin, J. F., (34) Mal.)
——, ——, LaForge, R. A. & Campbell, B. K., (34) (Mal.)
——, Sommers, A. H., Kerwin, J. F. & Campbell, B. K., (34) (Mal.)
Canaan, T., 85 (Am.), (719) (Lep.)
Cançado, J. R., 912 (Hel.)
Cánepa, C. H., with Pinedo & Fukelman, 532 (Def. Dis.)
Cannon, W. M., with Pratt-Thomas, 363 (Prot.)
Cantacuzène, A. & Lupasco, G., 529 (Hel.)
——, with Zotta, Radacovici, Lupasco & Teodoresco, 623 (Misc. Dis.)
Cantrell, W. & Jordan, H. B., 39 (Mal.)
Caplan, A. & Lindsay, J. K., 618 (Heat Str.)
Carbonell, M. & Contreras Dueñas, 592 (Lep.)
Carboni, E. A., with Fernandez, 595 (Lep.)
Card, W. I. & Walker, J. M., 707 (Typh.)
Carini, A., (716) (Am.)
Carlson, H. J., Bissell, H. D. & Mueller, M. G., 39 (Mal.)
——, Mueller, M. G. & Bissell, H. D., 174 (Mal.)
Carpenter, K. J., with Kodicek & Harris, 119 (Def. Dis.)
Carraud, G., (715) (Am.)
Carroll, D. G., with Winkenwerder, Hunninen, Harrison, Billings & Maier, 834 (Hel.)
Carroll, F. D., 1094 (Oph.)
Carruthers, L. B., 425 (Am.)
Cartwright, G. E. & Wintrobe, M. M., 608 (Haem.)
Caruana, M., with Durand & Benmussa, 56 (Leish.)
Casanova, J., 116 (Def. Dis.)
Casini, G., (274) (Mal.)
——, with Soper, Knipe, Riehl & Rubino, 877 (Mal.)
Casis Sacré, G., (1084) (Hel.)
Castellani, A., 1022 (Lab.)
Castillo, R. L., 276 (Mal.)
Castle, W. B., with Shen & Fleming, 1045 (Mal.)
Catanei, A., 536, 1019 (Der.)
——, with Parrot & Collignon, 700 (Mal.)
Causey, O. R., Deane, L. M. & Deane, M. P., 20 (Mal.)
——, with —— & Deane, M. P., 20 *bis* (Mal.)
Cawston, F. G., (221), (730), (1010) (Hel.)
Ceburkina, N. V., with Tovarnickij & Krontovskaja, (301) (Typh.)
Ceccaldi, J., 45 (Tryp.) 75 (Y.F.)
——, Paquier, P., Trinquier, E., Pelissier, A. & Vargues, R., 949 (Rab.)
Ceithaml, J. & Evans, E. A., Jr., 567 (Mal.)
Cerqueira, N. L. & Lane, J., 1063 (Y.F.)
——, with de Mendonça, 965 (Mal.)
Ceylon Health News, 238 (Misc. Dis.)
Chakraborty, D. C., with Sen & Basu, 209 (Chl.)
Chakravarty, H., with Chaudhuri, (522) (Am.)
Chakravarty, N. K., with Sen Gupta, P. C., (807) (Leish.)
Chala, J. I., 597 (Lep.)
Chandler, A. C., with Addis, (443) (Hel.)
Chang, C. T., with Chu, Huang & Kao, 423 (Chl.)
Chang, G. C. T., with Reimann, Chu, Liu & Ou, 423 (Chl.)
Chapman, N. B., Gibson, G. M. & Mann, F. G., (1037) (Mal.)
Chardome, M., with Peel, 339 (Hel.)
Chargaff, E., with Bendich, 407 (Typh.)
Charters, A. D., 620 (Ulc.)
Chatterjea, J. B., with Das Gupta, 745 (Haem.)
Chatterjee, H. N., 626 (Lab.), 998 (Chl.)
Chatterjee, P. K. & Banerjee, A., (989) (Typh.)
Chauderon, J., 342 (Def. Dis.)
Chaudhuri, A. K. R., (213) (Lep.)
Chaudhuri, R. N. & Chakravarty, H., (522) (Am.)
—— & Chauduri, M. N. R., 82 (Am.)
—— & Mukerji, A. K., 920 (Hel.)
Chauduri, M. N. R., with Chaudhuri, R. N., 82 (Am.)
Chaussinand, M., (1008) (Lep.)
Chelarescu, M., with Ciuca & Ballif, 561 (Mal.)
——, with ——, —— & Cristescu, 559 (Mal.)
Chen, G., with Schueler & Geiling, 1052 (Tryp.)
Cheney, G., with Bereston, 348 (Der.)
—— & Geib, W. A., 70 (Typh.)
Cheng Fang Tsu, 699 (Mal.)
Chenkin, T., with Brodie, Udenfriend & Dill, 793 (Mal.)
Chenoweth, B. M., Jr., with Stoll, Loughlin & Harris, 444 (Hel.)
——, with —— & Peck, 737 (Hel.)
Cherrington, M. E., with Darby & Ruffin, 456 (Sp.)
Chesterman, C. C., 282 (Mal.), 527 (Ys.), 683 (Reports, etc.), 1048 (Tryp.)
Chhatre, K. D., with Simeons, 77 (Pl.)

Chifflet, A. & Llopart, J., (835) (Hel.)
Chorine, V. & Tanguy, Y., 211 (Am.)
Chou, T. C., with Blaschko & Wajda, (698) (Mal.)
Choudhuri, R. K. S., 298 (Leish.)
Choussat, H., with Lebon & Duboucher, (525) (R.F.)
Christenson, L. D., with Oman, 953 (Mal.)
Christianson, H. B., Gordon, H. H., Daniels, W. B. & Lippincott, S. W., 24 (Mal.)
——, with ——, Dieuaide, Marble & Dahl, 963 (Mal.)
——, with —— & Lippincott, 176 (Mal.)
Christophers, S. R., 158 (Mal.)
Chu, L. W. & Huang, C. H., 423 (Chl.)
——, with ——, 419 (Pl.)
——, ——, Chang, C. T. & Kao, H. C., 423 (Chl.)
——, with Reimann, Chang, Liu & Ou, 423 (Chl.)
Chung, H. L., with Feng, 1055 (Leish.)
Ciaccio, G., with Giroud, (703) (Typh.)
Ciaravino, E. & Sofia, F., 495 (Mal.)
Cilli, V. & Corazzi, G., 846 (Vms.)
Ciuca, M., Ballif, L. & Chelarescu, M., 561 (Mal.)
——, ——, —— & Cristescu, A., 559 (Mal.)
Claessens, H., 49 (Tryp.)
Clapp, J. M., Fay, R. W. & Simmons, S. W., 563 (Mal.)
——, with —— & ——, 562 (Mal.)
Clark, W. B., 603 (Hel.)
Clark, W. M., 400 (Mal.)
Clarke, B. L. W., 231 (Def. Dis.)
Clarke, O., with Manson-Bahr, 456 (Sp.)
Claude, with Sarrouy & Combe, 55 (Leish.)
Cluer, E. H., 1004 (Am.)
Coates, A. E., 353 (Misc. Dis.)
Cochran, J. H., with Linduska & Morton, 681 (Ent.)
Cochrane, R. G., 1026 (B.R.), 1073, 1075 (Lep.)
——, Menon, K. P. & Pandit, C. G., 322 (Lep.)
—— & Ramanujain, K., 323 (Lep.)
Cocker, D. E., 257 (B.R.)
Coda, D., with Forbes, 158 (Mal.)
Codeleoncini, E., 302, 307, 512 (Typh.)
——, with d'Ignazio, 526 (R.F.), (193) (Typh.), 539 (Misc. Dis.)
Coggeshall, L. T., 278 (Mal.)
Coghill, N. F., Lawrence, J. & Ballantine, I D., 718 (R.F.)
Cohen, H. A., 756 (Lab.)
Cohen, M. G., with Hunter & Ingalls, 440 (Hel.)
Cohn, R., with Bianco, Saunders & Levine, 1034 (Mal.)
Coito, A. de M. F., with de Meira, 1014 (Hel.)
Colaço, A. T. F., 1020 (Ent.)
Cole, G., 602 (Hel.)
Cole, L. C. & Koepke, J. A., 468 (Ent.)
Collard, P. & Kendall, D., 903 (Am.)
Collignon, E., 701 (Mal.)
——, with Parrot & Catanei, 700 (Mal.)
——, with Sergent & Sergent, 692 (Mal.)
Collomb, H., with d'Ignazio, 178 (Mal.)
Combe, P., with Sarrouy, (319) (Am.)
——, with —— & Claude, 55 (Leish.)
Conejos, M., with Romaña & Lizondo, 300 (Leish.)
Congo Belge, 1024 (Reports, etc.)
Connellan, J. D., with Dakin, 915 (Hel.)
Conte, D., with Miyara, Horenstein & Córica, 815 (Pl.)
Contreras Dueñas, with Carbonell, 592 (Lep.)
Cook Islands, New Zealand, 1022 (Reports, etc.)
Cookson, L. O. C., 975 (Tryp.)
Corazzi, G., with Cilli, 846 (Vms.)
Corcoran, A. C. & Page, I. H., 539 (Misc. Dis.)
Córica, P., with Miyara, Conte & Horenstein, 815 (Pl.)
Corkill, N. L., with Kirk, 122 (Vms.)
Cornatzer, W. E., with Kyker & McEwen, 802 (Mal.)
Cortés, E., with Patiño Camargo, García, Santos, Zozaya, Rueda & Rosa del Valle, 1037 (Mal.)
Cortes, G., with Elderfield, Kremer, Kupchan & Birstein, (964) (Mal.)
Corvazier, R., with Boquet & Girard, 235 (Vms.)
da Costa, E., 558 (Mal.)
Costa, O. G. & Mariano, J., (726) (Lep.)
——, with Neves, 610 (Der.)
Coudert, J. & Baud, C., 241 (Ent.)
Coulston, F. & Huff, C. G., 970 (Mal.)
——, with ——, Laird & Porter, 1046 (Mal.)
Courtois, C., with Jepson & Moutia, 1030 (Mal.)
Coutinho, H., with Monteiro, Janz & de Loureiro, 340 (Def. Dis.)
Coutinho, J. O., 789 *bis* (Mal.)
Cova-Garcia, P., 630 (B.R.)
Covaleda, J., wth Matilla & Aparício Garrido, 279 (Mal.), 443 (Hel.), (608) (Haem.)
Covell, G., (155), 797 (Mal.)
Cowie, D. B., with Bartter, Most, Ness & Forbush, 1012 (Hel.)
Cowper, S. G., 217 (Hel.), 409 (Typh.)
Cox, G. W., 198 (Typh.)
Cox, H. R., with de Boer, Kunz & Koprowski, 712 (Den.)
——, with Koprowski, 518 (Den.)
——, Tesar, W. C. & Irons, J. V., 710 (Typh.)
Cram, E. B. & Files, V. S., 217 (Hel.)
de Crisenoy, J., (555) (Mal.)
Cristescu, A., with Ciuca, Ballif & Chelarescu, 559 (Mal.)
Cristol, S. J., Haller, H. L. & Lindquist, A. W., 466 (Ent.)
Crodel, B., with Haagen, 196 (Typh.)
Cropper, C. F. J., 88 (Am.)
Cross, J. B., with Scott, (447) (Hel.)
Culbertson, J. T., with Gellhorn & Rose, 602 (Hel.)
—— & Pearce, E., 229 (Hel.)
——, Rose, H. M., Hernández Morales, F., Oliver González, J. & Pratt, C. K., 924 (Hel.)
——, —— & Oliver-Gonzalez, J., 912 (Hel.)
Culwell, W. B., with Josephson & Trembley, 1030 (Mal.)
Culwick, A. T. & Fairbairn, H., 821 (R.F.)
——, with ——, 649, 1051 (Tryp.)
Cunha, with Fonseca, Cambournac, Pinto & Pereira, 487 (Mal.)

da Cunha, J. F., with Taylor, 414 (Y.F.)
Cuocolo, R., with Planet & de Almeida, 90 (Am.)
Curbelo, A., with Albacete & Florin, (1071) (R.F.)
——, with Milanes, Rodriguez, Kouri & Spies, 452 (Sp.)
Curd, F. H. S., (648) (Mal.)
——, Davis, M. I., Hoggarth, E. & Rose, F. L., (964) (Mal.)
——, Landquist, J. K. & Rose, F. L., (964) (Mal.)
——, Landquist, J. K. & Rose, F. L., (699) (Mal.)
—— & Raison, C. G., (699) (Mal.)
——, —— & Rose, F. L., (1037) (Mal.)
—— & Rose, F. L., 283 (Mal.)
Curtis, A. C. & Grekin, J. N., 1092 (Der.)
Cury, A., 235 (Der.)
Custer, R. P., 697 (Mal.)
——, with Malamud & Haymaker, 1097 (Heat Str.)
Cutler, J. G., 1079 (Hel.)
Czaczkes, J. W., with Olitzki & Kuzenok, 195 (Typh.)

D.

Dack, S. & Moloshok, R. E., 662 (Am.)
Dahl, L. K., with Gordon, Dieuaide, Marble & Christianson, 963 (Mal.)
Dakin, W. P. H. & Connellan, J. D., 915 (Hel.)
Dambreville, C., with Bellerive, 16 (Mal.)
Dame, L. R., 964 (Mal.)
Dammin, G. J., with Weller, 98 (Hel.)
Daniels, W. B., with Christianson, Gordon & Lippincoott, 24 (Mal.)
——, with Mason, Paddock & Gordon, 732 (Hel.)
Dao Van Ty, with Brumpt, 184 (Mal.)
Darby, W. J., Cherrington, M. E. & Ruffin, J. M., 456 (Sp.)
——, with Jones & Warden, 740 (Sp.)
——, Kaser, M. M. & Jones, E., 608 (Sp.)
Darlow, H. M., 548 (Reports, etc.)
Dartevelle, E. with Schwetz, 825 *bis* (Hel.)
Das, N. N., with Panja, 999 (Chl.)
Das Gupta, C. R., 1104 (B.R.)
—— & Chatterjea, J. B., 745 (Haem.)
——, with Majumder, 183 (Mal.)
——, with Napier, 1104 (B.R.)
Dauzier, M., 805 (Tryp.)
Davalos, A., with Beltrán, 960 (Mal.)
——, with —— & Sandoval, 968 (Mal.)
Davey, D. G., 282, 646, (648), 954 (Mal), 1048 (Tryp.)
——, with Butler & Spinks, 1037 (Mal.)
Davey, J. B., 683 (Reports, etc.)
Davey, P., with Hunt, 694 (Mal.)
Davey, T. F., 325 (Lep.)
David, W. A. L. & Bracey, P., 133, 544 (Ent.)
Davidson, L. S. P., (1092) (Haem.)
—— & Girdwood, R. H., 342 (Haem.), 741 (Sp.)
——, —— & Innes, E. M., 740 (Sp.)
Davies, G. E. & Fishburn, A. G., 369 (Lab.)
Davies, J. N. P., (238), 1098 (Misc. Dis.)
Davis, D. E., 705 (Typh.)
—— & Pollard, M., 303 (Typh.)
——, with —— & Olsen, 303 (Typh.)
Davis, D. H. S., 76 (Pl.)
Davis, F. P., with Haas, Wilcox & Ewing, 290 (Mal.)
Davis, L. J., (532) (Haem.)
Davis, M. I., with Curd, Hoggarth & Rose, (964) (Mal.)
Davis, W. A., 989 (Typh.)
——, with Soper, Markham & Riehl, 892 (Typh.)
Dawes, B., 762 (B.R.)
Deane, H. W., 17 (Mal.)
Deane, L. M., Causey, O. R. & Deane, M. P., 20 *bis* (Mal.)
Deane, M. P., with Deane, L. M. & Causey, 20 *bis* (Mal.)
Dearborn, E. H., 403 (Mal.)
—— & Marshall, E. K., Jr., 403, 568 (Mal.)
——, with Marshall, 292 *bis* (Mal.)
Debono, J. E., 985 (Leish.)
Deibel, H. K., 727 (Lep.)
Delage, B., with Blanc, 931 (Vms.)
De Lamater, J. N. & Hallman, F. A., 1066 (Am.)
Delpy, L. P., (755), (1020) (Ent.)
Delightish, J., with Gurevitch, 581 (Am.)
Dell, J. M., Jr. & Klinefelter, H. F., Jr., 171 (Mal.)
Del Sel, M. & Giberti, C., (665) (R.F.)
Denhoff, E., 598 (Hel.)
—— & Kolnodny, M. H., 748 (Ulc.)
Denny-Brown, D., 843 (Def. Dis.)
Deonier, C. C., Jones, H. A. & Incho, H. H., 289 (Mal.)
——, with —— & ——, 504 (Mal.)
Derham, A. P., (251) (Reports, etc.)
Derrick, E. H., 757 (Reports, etc.)
Desai, S. D., with Figueredo, 214 *bis* (Lep.)
Destaing, F., with Benhamou, Leonardon & Vargues, (586) (R.F.)
Dews, S. C. & Morrill, A. W., Jr., 134 (Ent.)
Dexter, M., with Bernstein, Stearns & Lott, (964) (Mal.)
Dey, N. C., with Panja & Ghosh, 1018 (Der.)
Dharmendra, Bose, R. & Sen Gupta, P. C., 189 (Leish.)
—— & Mukherji, N., (728) (Lep.)
—— & Santra, I., 324 (Lep.)
—— & Sen, N. R., 324, (430) (Lep.)
Dias, E., Laranja, F. S. & Nobrega, G., (651) (Tryp.)
Diaz, A. F., with Bozicevich, Donovan, Mazzotti & Padilla, 669 (Hel.)
Diaz-Rivera, R. S., Santos, J. J. & Perez-Santiago, E., 199 (Typh.)
Dick, G. W. A., 849 (Misc. Dis.)
—— & Bowles, R. V., 648 (Mal.)
Dickmann, G. H., 221 (Hel.)
Didier, R., (652) (Leish.)
Diercks, F. H. & Tibbs, R. O., 894 (Typh.)
Dieuaide, F. R., with Gordon, Marble, Christianson & Dahl, 963 (Mal.)
Diez Melchor, F., with Piedrola Gil, 636 (Rab.)
Dill, W., with Brodie & Udenfriend, 793 (Mal.)
——, with ——, —— & Chenkin, 793 (Mal.)
——, with ——, —— & Downing, 793 (Mal.)
Dimson, S. B. & McMartin, R. B., 32 (Mal.)
Dingle, J. H., 67 *bis*, 68, 69 (Typh.)

Discombe, G. & Watkinson, G., (608) (Haem.)
Dixon, K., with Drew & Samuel, 453 (Sp.)
Długosz., H., 278 (Mal.)
Dobrotvorskaya, N. V., with Kojevnikov & Latyshev, 940 (B.R.)
Dogiel, V. A., 477 (B.R.)
Dolmatova, A. V., 887, 982 (Leish.)
Dominguez, M., with Sabalette & Iglesias, 664 (R.F.)
Donaldson, A. W. & Otto, G. F., (335) (Hel.)
Donovan, A., with Bozicevich, Mazzotti, Diaz & Padilla, 669 (Hel.)
Do Prado, F. C. & Figliolini, F., 583 (Am.)
Dorofejew, W. N., with Stein, (214) (Lep.)
Dorrance, G. M. & Bransfield, J. W., 602 (Hel.)
Dostrovsky, A. & Sagher, F., (353) (Ulc.), 987, 1056 (Leish.)
Doucet, G., 451 (Def. Dis.)
——, with Neujean, 57 (Typh.)
Dougan, A. A., with Fletcher & Sammon, (209) (Am.)
Douglas, J. R., 755 (Ent.)
Down, H. A., with Harris, 581 (Am.)
Downing, G., with Brodie, Udenfriend & Dill, 793 (Mal.)
Downs, W. G., 1045 (Mal.)
——, Harper, P. A. & Lisansky, E. T., 953 (Mal.)
Drew, R., Dixon, K. & Samuel, E., 453 (Sp.)
Drummel, G., 840 (Def. Dis.)
Duarte, E., (676) (Der.)
Dubin, I. N., 955 (Mal.)
Dubois, A., 229 (Hel.), (806) (Tryp.)
Duboucher, G., with Lebon & Choussat, (525) (R.F.)
Dubourg, E., with Bonnin & Moretti, 343 (Haem.)
Duesselmann, W., with Wharton, 1099 (Misc. Dis.)
Duffner, G. J., 345 (Der.)
Dungal, N., 100 (Hel.)
Dunitz, J. D., with Fulton, 988 (Leish.)
Dunlop, E. E., 353 (Misc.)
Duport, M & Teodorescu, A. M., 996 (Den.)
Durand, P., Benmussa, S. & Caruana, M., 56 (Leish.)
Durand-Delacre, R., with Parrot, (754) (Ent.)
Durieux, C., 1023 (Reports, etc.)
——, Boiron, H. & Koerber, R., 991 (Y.F.)
Dwinelle, J. H., 907 (Ys.)
Dwyer, J. M., 1093 (Oph.)
van Dyke, H. B. & Gellhorn, A., 512 (Leish.)
——, with ——, 298 (Mal.)
——, with ——, & Tupikova, 334 (Hel.)

E.

Eads, R. B., 36 (Mal.), (136) (Ent.)
Earle, K. V., 53 (Tryp.), (129), 362 (Misc. Dis.)
Ecker, H. D. & Lubitz, J. M., (986) (Leish.)
Eddey, H. H., (250) (Reports, etc.)
Eddey, L. G., 879 (Mal.)
Edge, P. G., 862 (B.R.)
Edney, E. B., 1103 (Ent.)
Edwards, M. L., 715 (Am.)
Eeraerts, W., 49 (Tryp.)
Eichbaum, F. W., (930) (Vms.)
Einhorn, N. H., 695 (Mal.)
—— & Miller, J F., 215 (Hel.)
—— & Tomlinson, W. J., 393 (Mal.)
Eisenhower, E. W., with Russell & Roose, 425 (Am.)
Ekzempljarskaja, E. V., 907 (R.F.)
Ekzemplarskaya, E. V., 182 (Mal.)
El Ayadi, M. S., 433 (Hel.)
Elderfield, R. C., Kremer, C. B., Kupchan, S. M., Birstein, O. & Cortes, G., (964) (Mal.)
Elford, W. J., with van den Ende, Stuart-Harris, Fulton, Niven, Andrewes, Begg, White, Hawley, Mills, Hamilton & Thomas, 472 (B.R.)
El-Gammal, Y., with Attiah & Salem, 618 (Oph.)
Elishewitz, H., 1064 (Pl.)
El Kordy, M. I., 601 (Hel.)
——, with Ayadi, (312) (Am.)
——, with Halawani, 312 (Am.)
Ellerbrook, L. D., with Gordon, Marble, Lippincott & Hesselbrock, 173 (Mal.)
——, with Lippincott, Marble, Hesselbrock, Engstrom & Gordon, 166 (Mal.)
——, with ——, Paddock, Rhees & Hesselbrock, 601 (Hel.)
Elliott, M. E., with Burrows, Mather & Wagner, 422 (Chl.)
Ellis, J. M., with Young & Stubbs, 492 (Mal.)
——, with ——, ——, Burgess & Eyles, 275 (Mal.)
Elmendorf, J. E., Jr., with Barnhill, K. G. & Takos, M., 882 (Mal.)
——, Marucci, P. E., Griffin, J. B., Meyer, S. L. & Ryan, G. S., 466 (Ent.)
Elsdon-Dew, R., with Horner, R. & Cameron, D. A., 310 (Am.)
Elvehjem, C. A., with Luckey, Moore & Hart, 119 (Def. Dis.)
Emilio Caset, I., with Fernández Luna & Abbate, (1093) (Der.)
Emmons, C. W., with Olson & Bell, 848 (Der.)
Engstrom, W. W., Gordon, H. H., Marble, A. & Brunsting, H. A., 961 (Mal.)
——, with Lippincott, Marble, Ellerbrook, Hesselbrock & Gordon, 166 (Mal.)
Ephrati, P., 930 (Vms.)
Epidemiological Information Bull. (UNRRA Health Division), Wash., 73 (Y.F.)
Erfan, M., 913 (Hel.)
Erlanger, H., with Levine, 395 (Mal.)
Ermakova, N. E., 726 (Lep.)
Eskin, V. A., with Lisova & Sofiyev, 876 (Mal.)
Espersen, T., 100 (Hel.)
Etherington, D., Hickling, S., Roden, A. T. & Sellick, G., 163 (Mal.)
—— & Sellick, G., 19 (Mal)
Evans, E. A., Jr., 400 (Mal.)
——, with Ceithaml, 567 (Mal.)
Ewing, F. M., with Haas & Wilcox, 1045 (Mal.)
——, with ——, —— & Davis, 290 (Mal.)
Eyles, D. E., Hunter, G. W. & Warren, V. G., 837 (Hel.)
—— & Most, H., 838 (Hel.)
——, with Young, Stubbs, Ellis & Burgess, 275 (Mal.)

F.

Fabregoule, M., with Lebon, (665) (R.F.)
Fagan, K. J., 353 (Misc. Dis.)
Faget, G. H., 588, 593, 727 (Lep.)
—— & Pogge, R. C., (728) (Lep.)
——, —— & ——, 326 *bis* (Lep.)
——, ——, ——, Fite, G. L., Prejean, B. M. & Gemar, F., 594 (Lep.)
Fåhraeus, J., with Hammarström & Hellsten, 194, 652 (Typh.)
Faiguenbaum, J., with Neghme, 734 (Hel.)
Fain, A., 1080 (Hel.)
Fairbairn, H. & Burtt, E., 506 (Tryp.)
—— & Culwick, A. T., 649, 1051 (Tryp.)
——, with ——, 821 (R.F.)
Fairley, N. H., 175, 282, 398, 399 (Mal.), 538 (Misc. Dis.)
Fajerstein, S. G. & Besdenjesznich, K. V., 409 (Typh.)
Falcon Trejo, A., 274 (Mal.), 984 (Leish.)
Falcone, G., 558 (Mal.)
Fallander, S. R., with Hurlbut, Maple, Wilson & Husman, 881 (Mal.)
Fallis, A. M., (42) (Mal.)
Farooq, M. & Qutubuddin, M., 669 (Hel.)
Farr, A. G., with Young & McKendrick, (426) (R.F.), 448 (Hel.)
Faust, E. C., 833 (Hel.)
——, Heilbrunn, I., Lewis, R. & Murray, M. L., 1067 (Am.)
——, Hess, A. D. & Young, M. D., (158) (Mal.)
—— & Ingalls, J. W., with See, J. K., 331 (Hel.)
——, with Wright, McMullen & Bauman, 832 (Hel.)
Fawcett, D. G., with Arnold & Simmons, 277 (Mal.)
Fawdry, A. L., 344 (Haem.)
Fawzy, R. M., with Makar, 1011 (Hel.)
Fay, R. W., with Clapp & Simmons, S. W. 563 (Mal.)
——, Simmons, S. W. & Clapp, J. M., 562 (Mal.)
Federation Proc., 400 (Mal.)
Federico Guillot, C. & Osvaldo Curci, A., 591 (Lep.)
Federico Heinert, J., 1080 (Hel.)
Fedorov, V. G., 544 (Ent.)
Fehily, L., 673 (Def. Dis.)
Feigenbaum, A. & Landau, J., (618) (Oph.)
Feinstein, M., Yesner, R. & Marks, J. L., 66 (Typh.)
Feldman, A., 297 (Leish.)
Feng, L. C. & Chung, H. L., 1055 (Leish.)
Fenner, F., 514 (Typh.)
Ferebee, S. H. & Furcolow, M. L., 932 (Der.)
Ferguson, F. F., Arnold, E. H. & Upholt, W. M., 645 (Mal.)
——, with Mathis & Simmons, 644 (Mal.)
Ferguson, M. S., with Bang, Hairston & Graham, 435 (Hel.)
——, Graham, O. H., Bang, F. B. & Hairston, N. G., 435 (Hel.)
——, with Sullivan, 435 (Hel.)
Fermont, D. A., 874 (Mal.)
Fernandes, J. S., with Payne & Balthazar, 502 (Mal.)
Fernández, E., (308) (Y.F.)
Fernandez, J. M. M. & Carboni, E. A., 595 (Lep.)
Fernández Luna, D., Emilio Caset, I. & Abbate, E. A., (1093) (Der.)
Ferrebee, J. W. & Geiman, Q. M., 17 (Mal.)
——, Gibson, J. G. & Peacock, W. C., 17 (Mal.)
Ferreira, F. S. da C., 1052 (Tryp.)
Ferreira, L. de C., with Laemmert & Taylor, 414 (Y.F.)
Ferreira, M. O., with Rachou, 1032 (Mal.)
Ferro, R., with Jaffé, 433 (Hel.)
Ferro-Luzzi, G., with Jannone & Mara, 251 (Reports, etc.)
Fettes, D., 128 (Misc. Dis.)
Figliolini, F., with Do Prado, 583 (Am.)
Figueredo, N. & Desai, S. D., 214 *bis* (Lep.)
Files, V. S., with Cram, 217 (Hel.)
Findlay, G. M., 527 (Ys.), 579 (Y.F.)
——, Hardwicke, J. & Phelps, A. J., 406 (Tryp.)
——, with Hill & Macpherson, 94 (Ys.)
——, Maegraith, B. G., Markson, J. L. & Holden, J. R., 501 (Mal.)
—— & Markson, J. L., 884 (Bl.)
——, with Robertson, 747 (Haem.)
——, Robertson, W. M. & Zacharias, F. J., 344 (Haem.)
Fine, R., with Machella & Burgoon, 557 (Mal.)
Finland, M. & Lesses, M. F., 1061 (Typh.)
Fischer, H. & Almasy, F., (175) (Mal.)
Fischer, I. (320) (R.F.)
Fischer, W. J. H., Jr., & York, C. L. 25 (Mal.)
Fishburn, A. G., with Davies, 369 (Lab.)
Fisk, F. W., with Tarzwell, 563 (Mal.)
Fite, G. L., Cambre, P. J. & Turner, M. H., 1008 (Lep.)
——, with Faget, Pogge, Johansen, Prejean & Gemar, 594 (Lep.)
Fitzgerald, P. J., 274 (Mal.)
Flandrin, J., with Provost, (1006) (R.F.)
Fleck, L., 1057 (Typh.)
Fleming, E. M., with Shen & Castle, 1045 (Mal.)
Fleming, J., Bignall, J. R. & Blades, A. N., 711 (Den.)
Fletcher, D. E. & Rigdon, R. H. 42 (Mal.)
Fletcher, J. P., Dougan, A. A. & Sammon, G. K., (209) (Am.)
Fletcher, O. K., Jr., 392 (Mal.)
Flinn, L. B., Howard, J. W., Todd, C. W. & Scott, E. G., 413 (Typh.)
Floch, H., 53 (Tryp.), 103 (Hel.), 242 *bis* (Ent.), 486 (Mal.)
—— & Abonnenc, E., (131), (132), (681), (Ent.), (644) (Mal.)
—— & Grimaldi, J. T., 665 (R.F.)
—— & de Lajudie, P., 322, 589, 724, (727) (Lep.), 922 *bis* (Hel.), 1007 (Ys.), (1054) (Tryp.)
——, Taillefer-Grimaldi, J. & de Lajudie, P., 93 (R.F.)
Florin, M. C., with Albacete & Curbelo, (1071) (R.F.)
Florio, L., with Black & Stewart, 899 (Den.)
—— & Stewart, M. O., (711) (Den.)
Foner, A., with Adler, 313 (Am.)

Fonseca, F., Cambournac, F. J. C., Pinto, M. R., Pereira, J. M. & Cunha, A., 487 (Mal.)
da Fonseca, O., Jr., 610 (Der.)
da Fonseca Santos, H. S., 506 (Bl.)
Fontan, A., Verger, P., Mouliès, A. & Martin, C., 638 (Mal.)
Forbes, L. S. D. & Coda, D., 158 (Mal.)
Forbush, S., with Bartter, Cowie, Most & Ness, 1012 (Hel.)
Forrester, J. S., with Sayen, Pond & Wood, 305 (Typh.)
Forster, E. B., (100) (Hel.)
Fort & Zottner, 298 (Leish.)
Foshay, L., 837 (Hel.)
Foster, L. E., with Baker, Sommer, Meyer & Meyer, 712 (Pl.)
Fourman, L. P. R., 1087 (Sp.)
——, with Black & Bound, 1085 (Sp.)
Fowler, A. F., 978 (Tryp.)
Fowler, R., with Mezey, (350) (Der.)
Fox, D. G. R., with Yofe, 160 (Mal.)
Fox, I., (938) *bis* (Ent.)
Fox, J. P., 1062 (Y.F.)
—— & Laemmert, H W., Jr., 1062 (Y.F.)
——, with Peterson, 813 (Typh.)
Franks, M. B., 107, 446 (Hel.)
Fraser, P., 1022 (Reports, etc.)
Freed, J. E., with Burrows & Morehouse, 926 (Hel.)
Freedman, B., with Thomson, 673 (Def. Dis.)
Freeman, N. E., with Hodges, 621 (Misc. Dis.)
Freire Muñoz, C., 635 (Rab.)
de Freitas, G., with Muniz, 296 (Leish.), 571 (Tryp.)
French, R. S., with Meleney, 410 (Typh.)
Friedheim, E. A. H. & Vogel, H. J., (806) (Tryp.)
Friess, 442 (Hel.)
Frisch, A. W., with Oppenheim & Whims, 449 (Hel.)
Fritz, R. F., with Bradley, 180 (Mal.)
Frizzi, G., (1102) (Ent.)
Frommeyer, W. B., Jr. & Spies, T. D., 746 (Haem.)
Fukelman, R., with Pinedo & Cánepa, 532 (Def. Dis.)
Fuller, H. S., with Millspaugh, 705 (Typh.)
Fulton, F., with Henderson-Begg, 577 (Typh.)
——, with van den Ende, Stuart-Harris, Niven, Andrewes, Begg, Elford, White, Hawley, Mills, Hamilton & Thomas, 472 (B.R.)
Fulton, J. D. & Dunitz, J. D., 988 (Leish.)
——, with Harrison, 190 (Leish.)
—— & Lourie, E. M., 403 (Tryp.)
Funder, J. F. & Jackson, A. V., 204 (Typh.)
Furcolow, M. L., with Ferebee, 932 (Der.)
Furlonger, E., with Macpherson & Lee, 934 (Heat Str.)
Furtado, J. J., (322) (Lep.)
Fusté, R., with Kouri & Iriondo, (903) (Am.)

G.

Gabathuler, A. W., with Gabathuler, 924 (Hel.)
Gabathuler, M. J. & Gabathuler, A. W., 924 (Hel.)
Gage, J. C. & Rose, F. L., 500 (Mal.)
Gahan, J B. & Payne, G. C., 880 (Mal.)
Gajardo-Tobar, R. & Honorato, A., (852) (Misc. Dis.)
—— & Vildosola, E., 847 (Vms.)
Gale, G. W., 856 (Reports, etc.)
Gall, E. A. & Steinberg, A., 876 (Mal.)
Gallais, P., (877) (Mal.)
Gallenson, N., 413 (Typh.)
Galliard, H., 1014, 1082 (Hel.)
—— & Ngu, D. V., (695) (Mal.), 733, (737) (Hel.)
Gallup, B. N., with Smith & Cain, 177 (Mal.)
Gallut, J., 421, 660 (Chl.)
Garcia, E. Y., with Pesigan, 244 (Lab.)
García, V., with Patiño Camargo, Cortés, Santos, Zozaya, Rueda & Rosa del Valle, 1037 (Mal.)
Garcia Miranda, A., 431 (Lep.)
Garcin, G., 617 (Oph.)
Gardner, A. D., 426 (R.F.)
Garnham, P. C. C., 241 (Ent.), 666 (R.F.), 882 (Mal.)
—— & McMahon, J. P., 1084 (Hel.)
Gartrell, F. E. & Orgain, H., 20 (Mal.)
Gast Galvis, A., 848 (Der.)
——, with Anderson, 813 (Y.F.)
Gauld, R. L., with Robbins & Warner, 63 (Typh.)
Gauthier, A., 1094 (Oph.)
Gear, J. H. S., 1024 (Reports, etc.)
—— & Murray, N. L., 702 (Typh.)
Gee, F. L., 649 (Tryp.)
Geib, W. A., with Cheney, 70 (Typh.)
Geiling, E. M. K., with Schueler & Chen, 1052 (Tryp.)
Geiman, Q. M., Anfinsen, C. B., McKee, R. W., Ormsbee, R. A. & Ball, E. G., 1043 (Mal.)
——, with ——, ——, —— & ——, 1043 (Mal.)
——, with Ferrebee, 17 (Mal.)
——, with McKee, 649 (Mal.)
——, with ——, Ormsbee, Anfinsen & Ball, 1043 (Mal.)
Gelbjerg-Hansen, G., (754) (Misc. Dis.)
Gelfand, M., 232 (Def. Dis.), (462), 539, 937 (Misc. Dis.)
Geller, E. R., 927 (Hel.)
Gellhorn, A. & van Dyke, H. B., 298 (Mal.)
——, with ——, 512 (Leish.)
——, Rose, H. M. & Culbertson, J. T., 602 (Hel.)
——, Tupikova, N. A. & van Dyke, H. B., 334 (Hel.)
Gemar, F., with Faget, Pogge, Johansen, Fite & Prejean, 594 (Lep.)
Gendel, B. R., 609 (Haem.)
Gérard, R., (737) (Hel.)
Gerdjikoff, I., (789) (Mal.)
Gerin, L., (495) (Mal.)
Gerjovich, H. J., with Pijoan, Hopwood, Jachowski & Romine, 369 (Ent.)
Gersdorff, W. A. & Barthel, W. F., 467 (Ent.)
Gersh, I., with Tullis, Jenney, McLimans & Vinson, 895 (Typh.)
Gevorkov, A. A., 486 (Mal.)
Geyer, A., with Grall, (129) (Misc. Dis.)
Ghosh, A. R., with White, 799 (Mal.)
——, with —— & Rao, 37 (Mal.)

Ghosh, L. M., with Panja & Dey, 1018 (Der.)
Ghosh, N. C., with Banerjee, 452 (Def. Dis.)
Ghosh, S. K., with Panja, 998 (Chl.)
Ghosh, S. M., with Wu, McClymont & Roy, 464 (Ent.)
Ghyoot, E., 128 (Misc. Dis.)
Gbibs, A. J., (624) (Prot.)
Giberti, C., with Del Sel, (665) (R.F.)
Gibson, G. M., with Chapman & Mann, (1037) (Mal.)
Gibson, J. G., with Ferrebee & Peacock, 17 (Mal.)
Gibson, Q. H. & Harrison, D. C., 121 (Haem.)
Giglioli, G., (181), 504 (Mal.)
Gil, J., with Romaña, 405 (Tryp.)
——, with —— & de Romaña, 404 (Tryp.)
Gil Yepez, C., with Ignacio Baldo, Mayer & Pifano, 1077 (Hel.)
Gill, S., (190) (Leish.), 754 (Ent.)
Gillespie, J. B., with Sweigert & Turner, 460 (Der.)
Gillet, J. & Wanson, M., 132 (Ent.)
Gillett, J. D., with Haddow & Highton, 516 (Y.F.)
Gilliam, A. G., 408 (Typh.)
Gillman, J. & Gillman, T., (532) (Def. Dis.)
——, with ——, 118 (Def. Dis.)
Gillman, T. & Gillman, J., 118 (Def. Dis.)
Ginés, A. R. & Poletti, J. G., 528 (Lep.)
Gingrich, W. D. & Schoch, E. W., 970 (Mal.)
——, Schoch, E. W., Schwab, M. & Shepherd, C. C., 803 (Mal.)
Ginsburg, J. M. (681), 938 (Ent.)
Girard, G., 520 (Pl.)
——, with Ramon & Richou, 900 (Pl.)
—— & Sandor, G., 900 (Pl.)
Girard, O., with Boquet & Corvazier, 235 (Vms.)
Girardi, V. C. & Khoury, C., 125 (Der.)
Girdwood, R. H., with Davidson, 342 (Haem.), 741 (Sp.)
——, with —— & Innes, 740 (Sp.)
Girgis, B., with Bedford & Aidaros, 433 (Hel.)
Giroud, P., 304, 704, (704) (Typh.)
—— & Ciaccio, G., (703) (Typh.)
—— & Jadin, J., 891 (Typh.)
—— & Jude, A., 1057 (Typh.)
——, with Montel, 322 (Lep.)
Giroux, J., with Harant & Braun-Blanquet, 1013 (Hel.)
Glagoleva, E. M., (692) (Mal.)
Glasgow, J. P., 47 (Tryp.)
Glenn, P. M., Kaplan, L. I., Read, H. S. & Becker, F. T., 167 (Mal.)
Godfrey, L. W. (443) (Hel.)
Gokhale, V. G., with Pendse, Phalnikar & Bhide, (681) (Ent.)
Golberg, L., with de Meillon, 525 (R.F.)
Gold, E. M., with Wright, 224 (Hel.)
Goldblum, N., with Birnbaum & Kligler, 22 (Mal.)
Golden, A., 678 (Ulc.)
Goldsmith, G. A., 674 (Haem.)
Goldsmith, K., 502 (Mal.)
Goldstein, A. C., 328 (Hel.)
Goldstein, L., with Grasset, (1017) (Vms.)
Gómez Marcano, A., 390 (Mal.)
Gomez Rodríguez, G., 137 (Lab.)
Gonçalves, A. P., 236, 349, 847 (Der.)
González Barrientos, G., with Hernández Morales, 228 (229) (Hel.)
González Prendes, M. A., with Ibarra Pérez, 430, 590 (Lep.)
——, with Oteiza Setien & Ibarra Perez, 95, 589 (Lep.)
Goodhue, L. D., (369) (Ent.)
Gordadze, G. N., with Kamalov, Tsutsunava, Khizanishvili, Kamalova & Buguianishvili, 224 (Hel.)
Gordon, E. S., with Albright, (714) (Am.)
Gordon, H. H., with Christianson, Daniels & Lippincott, 24 (Mal.)
——, —— & Lippincott, S.W., 176 (Mal.)
——, Dieuaide, F. R., Marble, A., Christianson, H. B. & Dahl, L. K., 963 (Mal.)
——, with Engstrom, Marble & Brunsting, 961 (Mal.)
——, with Lippincott, Marble, Ellerbrook, Hesselbrock & Engstrom, 166 (Mal.)
——, Marble, A., Lippincott, S. W., Hesselbrock, W. B. & Ellerbrook, L. D., 173 (Mal.)
——, with Mason, Daniels & Paddock, 732 (Hel.)
Gordon, I., 801 (Mal.) (855) (Ent.)
Gordon, J. E. & Knies, P. T., (359) (Pl.)
Gordon, R. M. & Hill, M. A., 402 (Mal.)
Gosselin, R. E., with Molnar, Towbin, Brown & Adolph, 461 (Heat Str.)
Goswell, G., (519) (Den.)
Gouck, H. K., 78 (Pl.)
Graham, O. H., with Bang, Hairston & Ferguson, 435 (Hel.)
——, with Ferguson, Bang & Hairston, 435 (Hel.)
——, with McMullen, 917 (Hel.)
Grainger, W. E., 644 (Mal.)
Grall, A. & Geyer, A. (129) (Misc. Dis.)
Grana, P. C., 130 (Misc. Dis.)
Granier-Doyeux, M. & Holz, S., (1092) (Haem.)
Grant, C. W., with McLimans, 706 (Typh.)
Grasset, E., 419, 520 (Pl.)
—— & Goldstein, L., (1017) (Vms.)
Graves, P. R., 605 (Def. Dis.)
Graziani, J. G., with Martin, Collins & Lincicum, 334 (Hel.)
Great Britain, 260 (B.R.)
Greenberg, M., Pellitteri, O. J. & Jellison, W. L., 990 (Typh.)
——, ——, Klein, I. F. & Huebner, R. J., 707 (Typh.)
——, with Shookhoff & Birnkrant, 603 (Hel.)
Greenwood, M., 636 (Rab.), 862 (B.R.)
Greif, R. L., 714 (Am.)
Grekin, J. N., with Curtis, 1092 (Der.)
Grenoilleau, G., (621) (Ulc.), 658 (Pl.)
Greval, S. D. S. & Bhaduri, P. N., 239 (Misc. Dis.)
Griffin, J. B., with Elmendorf, Marucci, Meyer & Ryan, 466 (Ent.)
Griffiths, J. T., Jr., 1060 (Typh.)
Grimaldi, J. T., with Floch, 665 (R.F.)
Groupe, V., Nigg, C. & Macfarlane, J. O., 197 (Typh.)
Guennnec, with Soubigou & Autheman, 585 (Am.)
Guha, P. C., with Bami & Iyer, (793) (Mal.)
—— & Mukherjee, S. P., (558) (Mal.)

Guillaume, A., (701) (Mal.)
Guimarães, F. N., 719 (Ys.)
Gunnison, J. B., with Lazarus, 1064 (Pl.)
Gunther, C. E. M., 178 (Mal.)
Gunther, F. A. & Tow, L. R., 133 (Ent.)
Gurevitch, J. & Delightish, J., 581 (Am.)
Guyane, Française, 53 (Tryp.), 103 (Hel.), 486 (Mal.), 242 *bis* (Ent.)
György, P., with Perlingiero, (622) (Misc. Dis.)

H.

Haagen, E. & Crodel, B., 196 (Typh.)
Haagn, E. 892 (Typh.)
Haas, V. H., Wilcox, A., Davis, F. P. & Ewing, F. M., 290 (Mal.)
——, —— & Ewin, F. M., (1045) (Mal.)
Habel, K., 951 (Rab.)
Habibi, A., with Parrot, (132) (Ent.)
Hackett, C. J., 527 *bis* (Ys.)
Hackett, L. W., 276 (Mal.)
——, with Putnam, 15 (Mal.)
Hackman, R. H., 1103 (Ent.)
Hadaway, A. B. & Barlow, F., 1038 (Mal.)
——, with ——, 1053 (Tryp.)
——, with Symes, 545 (Ent.)
Haddad, C., Sheiban, A. & Budeir, R., 320 (R.F.)
Haddow, A. J., Gillett, J. D. & Highton, R. B., 516 (Y.F.)
——, with Smithburn, 71 (Y.F.)
——, ——, Mahaffy, A. F. & Bugher, J. C., 992 (Y.F.)
Hafiz, A., with Halawani, 329 (Hel.)
Haggis, (1036) (Mal.)
Hair, H. C., with Peterkin, 643 (Mal.)
Hairston, N. G., with Bang, 435 (Hel.), 1038 (Mal.)
——, with ——, Graham & Ferguson, 435 (Hel.)
——, with ——, Maier & Trager, 281, 503 (Mal.)
——, with ——, Trager & Maier, 696 (Mal.)
——, with Ferguson, Graham & Bang, 435 (Hel.)
——, with Trager & Bang, 1036 (Mal.)
Halawani, A., 331, (667) (Hel.)
—— & Abdallah, A., 220 (Hel.)
——, Baz, I. & Morkos, F., 698 (Mal)
——, Baz, I. I. & Morcos, F., 792 (Mal.)
—— & El Kordy, M., 312 (Am.)
—— & Hafiz, A., 329 (Hel.)
——, Nor-el-Din, G., Shaker, M. & Khalek, F. A., 99 (Hel.)
Hall, G. A. M., 937 (Misc. Dis.)
Hall, T. F. & Hess, A. D. 1042 (Mal.)
—, with Penfound & Hess, 21 (Mal.)
Hallberg, V., 591 (Lep.)
Haller, H. L., with Cristol & Lindquist, 466 (Ent.)
Hallman, F. A., with DeLamater, 1066 (Am.)
Hamilton, F., with van den Ende, Stuart-Harris, Fulton, Niven, Andrewes, Begg, Elford, White, Hawley, Mills & Thomas, 472 (B.R.)
Hammarström, E., Fahraeus, J. & Hellsten, M.D.H., 194 (Typh.)
——, Hellsten, H. & Fåhraeus, J., 652 (Typh.)
Hammouda, M., 221 (Hel.)
Hampton, S. F., 573 (Typh.)
Handler, P., (739) (Def. Dis.)
Hanks, J. H., (213), 323, 721, 722 *bis*, 723 *bis*, 724 (Lep.)
Hansen, E. L., with Anderson & Johnstone, 819 (Am.)
——, with Bond, Bostick & Anderson, 585 (Am.)
Harant, H., Giroux, J. & Braun-Banquet, M., 1013 (Hel.)
Hardwicke, J., with Findlay & Phelps, 406 (Tryp.)
Hare, K. P., (621) (Ulc.), 738 (Def. Dis.)
Hargett, M. V., with Burruss, 994 (Y.F.)
Hargreaves, W. H., 80 (Am.)
Haridas, G., 844 (Def. Dis.)
Harper, J. O., 680 (Ent.)
Harper, P. A., with Downs & Lisansky, 953 (Mal.)
——, with Levine, 953 (Mal.)
——, Lisansky, E. T. & Sasse, B. E., 953 (Mal.)
Harris, A. H. & Down, H. A., 581 (Am.)
——, with Stoll, Loughlin & Chenoweth, 444 (Hel.)
Harris, J. O., (887) (Leish.)
Harris, L. J., with Kodicek & Carpenter, 119 (Def. Dis.)
Harrison, C. V. & Fulton, J. D., 190 (Leish.)
Harrison, D. C., with Gibson, 121 (Haem.)
Harrison, T., with Winkenwerder, Hunninen, Billings, Carroll & Maier, 834 (Hel.)
Hart, E. B., with Luckey, Moore & Elvehjem, 119 (Def. Dis.)
Hartz, P. H., 101 (Hel.)
——, with Van der Sar, (125) (Der.)
Harvey, A. E. C., 679 (Ent.)
Harvey, C., 353 (Misc. Dis.)
Hashem, M., 730 (Hel.)
Hatchett, S. P., 366 (Ent.)
Haulon, J., with Pasquet, 127 (Heat Str.)
Hauschka, T., Saxe, L. H., Jr. & Blair, M., 1054 (Tryp.)
Havard, R. E., with Maegraith, 44 (Bl.)
Hawking, F., 648, 970 (Mal.), 1054 (Tryp.)
Hawksley, J. C., 360 (Misc. Dis)
Hawley, W. L., with van den Ende, Stuart-Harris, Fulton, Niven, Andrewes, Begg, Elford, White, Mills, Hamilton & Thomas, 472 (B.R.)
Haymaker, W., with Malamud & Custer, 1097 (Heat Str.)
——, with Weil, 577 (Typh.)
Hayman, J. M., with Most, Kane, Lavietes, London & Schroeder, 496 (Mal.)
Hayman, J. M., Jr., with Bazemore, Johnson & Swanson, 559 (Mal.)
——, with Most & Wilson, 443 (Hel.)
Haynes, D. M. & Hess, W. I., 460 (Der.)
Hays, E. E. (533) (Haem.)
Hayward, G. W., 81 (Am.)
Hazra, A. K., Lahiri, D. C. & Sokhey, S. S., (235) (Vms.)
Heath, G. B. S. & Mitchell, J. G., 754 (Ent.)
Heath, R. F., (1039) (Mal.)
Heathcote, R. St. A., 130 (Misc. Dis.)
Hector Cambiaso, R., with Olle, 1095 (Oph.)
Hedge, K. V., with Shamanna, 520 (Pl.)
Hegsted, D. M., with Roos & Stare, 403 (Mal.)
Heide, E. A., 193 (Typh.)

Heidelberger, M., with Mayer, 172 (Mal.)
Heilbrunn, I., with Faust, Lewis & Murray, 1067 (Am.)
Heilig, R., 512 (Typh.)
Heinle, R. W., with Suárez, Welch, Suárez & Nelson, 607 (Sp.)
Heisch, R. B., 678 (Misc. Dis.)
Helbing, C. H., with Campbell & Kerwin, (34) (Mal.)
Hellerman, L., Bovarnick, M. R. & Porter, C. C., 400 (Mal.)
Hellsten, H., with Hammarström & Fåhraeus, 652 (Typh.)
Hellsten, M. D. H., with Hammarström & Fahraeus, 194 (Typh.)
Helwig, E. B., with Jennison & Milstone, (679) (Misc. Dis.)
Helwig, E. G. & Brown, R. G., (442) (Hel.)
Henderson, A. B. & Thornell, H. E., 609 (Haem.)
Henderson, C. R., with Kark, Aiton, Pease, Bean, Johnson & Richardson, 842 (Def. Dis.)
Henderson-Begg, A., 509 (Tryp.)
—— & Fulton, F., 577 (Typh.)
Henrard, C., with van Hoof & Peel, 979 (Tryp.)
——, Peel, E. & Wanson, M., 103 (Hel.)
Henry, A. F. X., 1033 (Mal.)
Henry, A. J., 989 (Leish.)
Henry, E., with Rodhain, (807) (Tryp.)
Henry, P. S. H. & Rees, W. H., 676 (Heat Str.)
Henry-Dustin, M.-J., (846) (Vms.)
Hentz, H. & Noel, G., (406) (Leish.)
Herbert, D., 713 (Pl.)
Herman, F., with Jadin, 390 (Mal.)
Hernández-Mora, C., (807) (Tryp.)
Hernández Morales, F., with Culbertson, Rose, Oliver González, J. & Pratt, 924 (Tryp.)
—— & González Barrientos, G., 228, (229) (Hel.)
——, F. & Maldonado, J. F., with Pratt, C. K., 330 *bis* (Hel.)
——, F., with Oliver González, 830 (Hel.)
——, —— & Pratt, C. K., 434 (Hel.)
——, Pratt, C. K. & Oliver González, J., 731 (Hel.)
——, with Santiago-Stevenson & Martinez, 902 (Am.)
——, Suárez, R. M. Pratt, C. K. & Oliver González, J., 218 (Hel.)
Hernando Ordóñez, J., 532 (Def. Dis.)
Hernberg, C. A., 15, 951 (Mal.)
Herriott, R. M., 133 (Ent.)
Hertzberg, R., 26 (Mal.)
Hess, A. D., with Faust & Young, (158) (Mal.)
——, with Hall, 1042 (Mal.)
—— & Keener, G. G., Jr., 880 (Mal.)
——, with Penfound & Hall, 21 (Mal.)
——, with Watson & Kiker, 801 (Mal.)
Hess, W. I., with Haynes, 460 (Der.)
Hesselbrock, W. B., with Gordon, Marble, Lippincott & Ellerbrook, 173 (Mal.)
——, with Lippincott, Marble, Ellerbrook, Engstrom & Gordon, 166 (Mal.)
——, with ——, Paddock, Rhees & Ellerbrook, 661 (Hel.)
Hewer, T. F., 527 (Ys.)
Hewitt, R. I., with Richardson, Seager, Brook, Martin & Maddux, 289 (Mal.)
Hibbs, R. E., 607 (Def. Dis.)
Hickling, S., with Etherington, Roden & Sellick, 163 (Mal.)
Hicks, J. D., 460 (Der.)
Higashi, A., with Welch, Peters, Bueding & Valk, 1015 (Hel.)
Highby, P. R., 446 (Hel.)
Highton, R. B., with Haddow & Gillett, 516 (Y.F.)
Hill, E. L. & Ingraham, S. C., 894 (Typh.)
Hill, I. G. W., 360 (Misc. Dis.)
Hill, K. R., 87, 901 (Am.)
——, Findlay, G. M. & Macpherson, A., 94 (Ys.)
Hill, M. A., with Gordon, 402 (Mal.)
Hills, A. G., 169 (Mal.)
Hilmy, I. S., with Khalil, 97 (Hel.)
Hilson, D., with Mirvish, 615 (Oph.)
Hindle, E., 795 (Mal.)
Hinman, E. H. & Baez, M. M., 371 (Reports, etc.)
Hird, A. J., (652) (Leish.)
Hitch, J. M., 921 (Hel.)
Hitchcock, D. J. & Rawson, G. W., 84 (Am.)
——, with ——, 716 (Am.)
Ho, E. A., 189 (Leish.)
——, Hsu, T. H. & Soong, T. H., 987 (Leish.)
Hoare, C. A., 1048 (Tryp.)
Hocking, K. S., 879, 1038 (Mal.)
Hodges, H. H. & Freemen, N. E., 621 (Misc. Dis.)
Hodgson, C. H., 578 (Bart.)
Hoekstra, J., with Snijders & Polak, 993 (Y.F.)
Hoffman, W. H., 517 (Y.F.)
Hoggarth, E., with Curd, Davis & Rose, (964) (Mal.)
Hogue, M. J., (1102) (Prot.)
Holden, J. R., with Findlay, Maegraith & Markson, 501 (Mal.)
Holland, T., 618 (Heat Str.)
Hollands, R. A. & Palmer, E. D., 439 (Hel.)
Hollenbeck, A. H., (241) (Ent.)
Holler, G. & Zajitschek, R., 318 (Am.), 1057 (Typh.)
Hollis, M. D., (182) (Mal.)
Holton, P., 835 (Hel.)
Holz, S., with Granier-Doyeux, (1092) (Haem.)
Honorato, A., with Gajardo-Tobar, (852) (Misc. Dis.)
Hood, A., 360 (Misc. Dis.)
Hood, M., (856) (Lab.)
van Hoof, 1024 (Reports, etc.)
——, Henrard, C. & Peel, E., 979 (Tryp.)
van Hoof, L. M. J. J., 1048 (Tryp.)
Hoogstraal, H., with King, 543 (Ent.)
Hooper, J. M., with Irons, 710 (Typh.)
Hopkins, D. L. & Warner, K. L., 83 (Am.)
Hopper, J., Jr., with Monroe, 246 (Lab.)
Hopwood, M. L, with Pijoan, Gerjovich, Jachowski & Romine, 369 (Ent)
Horenstein, B., with Miyara, Conte & Córica, 815 (Pl.)
van Horn, F., with Benedek, Lewe, Price & Koransky, 458 (Der.)
Horner, R., with Elsdon-Dew & Cameron, 310 (Am.)

Horton, R. K., with Westphal, 565 (Mal.)
Hovanitz, W., 566 (Mal.)
Howard, B., with Balamuth, 582 (Am.)
Howard, J. W., with Flinn, Todd & Scott, 413 (Typh.)
Howell, C. A. H., 929 (Sp.)
Howell, K. M., with Knoll, 524 (Am.)
Howell, T. H., 1017 (Sp.)
Hsu, K. C., 1008 (Hel.)
Hsu, T. H., with Ho & Soong, 987 (Leish.)
Hu, S. M. K., 1083 (Hel.)
Huang, C. H. & Chu, L. W., 419 (Pl.)
——, with ——, 423 (Chl.)
——, with ——, Chang & Kao, 423 (Chl.)
Huebner, R. J., 709 (Typh.)
——, with Greenberg, Pellitteri & Klein, 707 (Typh.)
——, Jellison, W. L. & Armstrong, C., 897 (Typh.)
——, —— & Pomerantz, C., 411 (Typh.)
——, Stamps, P. & Armstrong, C., 411 (Typh.)
——, with Topping & Shepard, 70 (Typh.)
Huff, C. G., 158 (Mal.)
——, with Coulston, 970 (Mal.)
——, ——, Laird, R. L. & Porter, R. J., 1046 (Mal.)
Hughes, A. C. C., 205 (Den.)
Hughes, C. O. & Brackett, S., 41 (Mal.)
——, with ——, Waletzky, Baker & Williams, 40 (Mal.)
Hughes, T. P., with Mahaffy & Smithburn, 72 (Y.F.)
——, with Perlowagora, 657 (Y.F.)
Hull, R., Lovell, B. J., Openshaw, H. T. & Todd, A. R., (642) (Mal.)
Humphreys, F. A. & Campbell, A. G., 758 (Reports, etc.)
Hunninen, A. V., with Winkenwerder, Harrison, Billings, Carroll & Maier, 834 (Hel.)
Hunt, R. & Davey, P., 694 (Mal.)
Hunter, G. W., with Eyles & Warren, 837 (Hel.)
——, Ingalls, J. W. & Cohen, M. G., 440 (Hel.)
Hunter, J. K., 661 (Dys.)
Hurlbut, H. S., Maple, J. D., Wilson, C. S., Fallander, S. R. & Husman, C. N., 881 (Mal.)
——, with Travis, Maple & Hurlbut, 646 (Mal.)
Husman, C. N., with Hurlbut, Maple, Wilson & Fallander, 881 (Mal.)
——, with Travis, Maple & Hurlbut, 656 (Mal.)
Hussain, A., with Sharma, 737 (Hel.)
Hutchison, W. C. & Kermack, W. O., (877) (Mal.)
Hynes, M., Ishaq, M. & Morris, T. L., 233 (Def. Dis.)
——, ——, Morris, T. L. & Verma, O. P., 744 (Haem.)
——, —— & Verma, O. P., 744 (Haem.)

I.

Ibarra Pérez, R. & González Prendes, M. A., 430, 590 (Lep.)
——, with Oteiza Setien & Gonzalez Prendes, 95, 589 (Lep.)
Ibrahim, K. G., 618 (Oph.)
Iglesias, R., with Sabalette & Dominguez, 664 (R.F.)
Ignacio Baldo, J., Gil Yepez, C., Mayer, M. & Pifano, C. F., 1077 (Hel.)
Ignacio Chala H., J., 430 (Lep.)
—— & Lleras Restrepo, F., 323 (Lep.)
d'Ignazio, C., 193 (Typh.), (525) (Am.)
—— & Codeleoncini, E., (193) (Typh.), 526 (R.F.), 539 (Misc. Dis.)
—— & Collomb, H., (178) (Mal.)
Ikejiani, O., 572 *quat.*, (885) (Tryp.)
—— & Mautner, L. S., 185 (Tryp.)
Incho, H. H., with Deonier & Jones, 289 (Mal.)
——, with Jones & Deonier, 504 (Mal.)
India, 252 (B.R.)
Indian Research Fund Association, 966 (Mal.), 997 (Pl.), 998 (Chl.)
Infante Gómez, A., with Moreno Berdugo, 320 (R.F.)
Ingalls, J. W., with Faust & See, 331 (Hel.)
——, with Hunter & Cohen, 440 (Hel.)
——, with McMullen, 917 (Hel.)
Ingalls, J. W., Jr., 442 (Hel.)
Ingraham, H. S. & Lapenta, R. G., 212 (R.F.)
Ingraham, S. C., with Hill, 894 (Typh.)
Innes, E. M., with Davidson & Girdwood, 740 (Sp.)
Innes, J. R. M., 877 (Mal.)
Internat. J. Leprosy, 327 (Lep.)
Internat. Labour Review, (144) (Reports, etc.), 327 (Lep.)
Iriarte, D. R., (626) (Ent.)
Iriondo, M., with Kourí & Fusté, (903) (Am.)
Irons, J. V., with Cox & Tesar, 710 (Typh.)
—— & Hooper, J. M., 710 (Typh.)
——, Murphy, J. N. & Wolfe, D. M., 710 (Typh.)
——, with Topping & Shepard,, 710 (Typh.)
Ishaq, M., with Hynes & Morris, 233 (Def. Dis.)
——, with ——, —— & Verma, 744 (Haem.)
——, with —— & Verma, 744 (Haem.)
Ivanova, L. V., 161, 488 (Mal.)
Iyengar, M. O. T., 564 (Mal.)
Iyengar, N. K., with Bose & Mukerji, 169 (Leish.)
Iyer, B. H., with Bami & Guha, (793) (Mal.)

J.

Jackowski, L. A., wieth Pijoan, Gerjovich, Hopwood & Romine, 369 (Ent.)
Jachowski, L. A., Jr. & ——, 368 (Ent.)
Jackson, A. V., with Funder, 204 (Typh.)
Jackson, C. H. N., 47 (Tryp.)
Jackson, E. B., with Smadel, Bennett & Rights, 513 (Typh.)
Jackson, H. & Oleesky, S., (321) (R.F.)
Jacoby, H., 88 (Am.), (754), 937 (Misc. Dis.)
Jacques, J. J. C., (1076) (Hel.)
Jadin, J., with Giroud, 891 (Typh.)
Jadin, L. B. & Herman, F., 390 (Mal.)
Jaffé, R. & Ferro, R. 433 (Hel.)
Jame, L., 178 (Mal.)
—— & Jude, A., (893) Typh.)

Jannone, G., Ferro-Luzzi, G. & Mara, L., 251 (Reports, etc.)
Janz, G. J., with Monteiro, Coutinho & de Loureiro, 340 (Def. Dis.)
Jellison, W. L., with Greenberg & Pellitteri, 990 (Typh.)
——, with Huebner & Armstrong, 897 (Typh.)
——, with —— & Pomerantz, 411 (Typh.)
Jenney, E., with Tullis, Gersh, McLimans & Vinson, 895 (Typh.)
Jennings, G. H., 278 (Mal.)
Jennison, D. B., Helwig, E. B. & Milstone, J. H., (679) (Misc. Dis.)
Jepson, W. F., Moutia, A. & Courtois, C., 1030 (Mal.)
Jerace, F., 808 (Leish.)
Jersild, M. & Meulengracht, E., 457 (Haem.)
Jettmar, H. M., 750 (Misc. Dis.)
Jhatakia, K. U., 129 (Misc. Dis.)
—— & Mankad, K. K., (663) (Am.)
Joannides, G., 627 (Lab.)
Johansen, F. A., with Faget & Pogge, 326 *bis* (Lep.)
——, with ——, ——, Fite, Prejean & Gemar, 594 (Lep.)
John, M. B., with Sharp, 349 (Der.)
Johnson, C. G., 545, 625 (Ent.)
Johnson, D. H., 200 (Typh.)
Johnson, H. A. & Yates, M. W., 1038 (Mal.)
Johnson, H. H., with Bazemore, Swanson & Hayman, 559 (Mal.)
Johnson, J. G., (1102) (Prot.)
Johnson, R. E., with Kark, Aiton, Pease, Bean, Henderson & Richardson, 842 (Def. Dis.)
Johnston, C. R. St., with Morrison, 742 (Sp.)
Johnstone, H. F., with Rice & Kearns, 544 (Ent.)
Johnstone, H. G., with Anderson & Hansen, 819 (Am.)
Johnstone, R. D. C., 284, 396, 697 (Mal.), 530 (Hel.)
Jolly, A. T. H., 348 (Der.)
Jones, B., 469 (Ent.)
Jones, E., with Darby & Kaser, 608 (Sp.)
——, Warden, H. F. & Darby, W. J., 740 (Sp.)
Jones, H. A., with Deonier & Incho, 289 (Mal.)
——, Incho, H. H. & Deonier, C. C., 504 (Mal.)
——, McCollough, G. T. & Morton, F. A., 467 (Ent.)
——, Titus, H. A. & Oberg, M. W., (467) (Ent.)
Jones, M. F. & Brady, F. J., 217 (Hel.)
Jones, W. R., 313, 1069 (Am.)
Jongco, A. P., with Tupas, 238 (Misc. Dis.)
Jope, E. M., 743 (Haem.)
Jordan, H. B., with Cantrell, 39 (Mal.)
Jordan, W. S., with Saunders & Bianco, 108 (Hel.)
Jörg, M. & Aguirre, J. A., 115 (Def. Dis.)
Joseph, A. D., 621 (Misc. Dis.)
Josephson, E. S., Culwell, W. B. & Trembley, H. L., 1030 (Mal.)
——, Udenfriend, S. & Brodie, B. B., 793 (Mal.)
J. Univ. Bombay, (666) (Hel.)
Jude, A. & Brumpt, L., 811 (Typh.)
Jude, A., with Giroud, 1057 (Typh.)
——, with Jame, (893) (Typh.)

K.

Kahn, R. L., with Wheeler, McDermott & Adler, (642) (Mal.)
Kaleita, E., with Becker & Burks, 177 (Mal.), 583 (Am.)
Kalfayan, B. H., 1006 (R.F.)
Kalra, S. L., 575 (Typh.)
Kamalov, N. G., 668 (Hel.)
——, Gordadze, G. N., Tsutsunava, T. N., Khizanishvili, A. O., Kamalova, A. G. & Buguianishvili, S. M., 224 (Hel.)
Kamalova, A. G., with Kamalov, Gordadze, Tsutsunava, Khizanishvili & Buguianishvili, 224 (Hel.)
Kane, C. A., with Most, Lavietes, London, Schroeder & Haymen, 496 (Mal.)
Kantor, F., with Ritchken, 1078 (Hel.)
Kao, H. C., with Chu, Huang & Chang, 423 (Chl.)
Kaplan, F. M., with Kessel, Moore & Schireson, 524 (Am.)
Kaplan, L. I., with Becker, Read & Boyd, 280 (Mal.)
——, with Glenn, Read & Becker, 167 (Mal.)
——, with Read & Becker, 177 (Mal.)
——, —— & ——, with Boyd, M. F., 25, 281 (Mal.)
Kark, R. M., Aiton, H. F., Pease, E. D., Bean, W. B., Henderson, C. R., Johnson, R. E. & Richardson, L. M., 842 (Def. Dis.)
Kark, W., 170 (Mal.)
Karmakar, G., with Rao, 1089 (Haem.)
Kartman, L., 420 (Pl.)
—— & da Silveira, M. M., 38 (Mal.)
Kaser, M. M., with Darby & Jones, E., 608 (Sp.)
Katzenellenbogen, I., 539 (Misc. Dis.)
Katzin, B. & Most, H., 440 (Hel.)
Kaump, D. H., (243) (Lab.)
Kauntze, W. H., 470 (Reports, etc.)
Kean, B. H., with Shrager, 170 (Mal.)
—— & Taylor, C. E., 166 (Mal.)
Kearns, C. W., with Rice & Johnstone, 544 (Ent.)
Keen, P., 662 (Am.)
Keener, G. G., Jr., with Hess, 880 (Mal.)
Kekwick, A., Wright, R. J. & Raper, A. B., (674) (Def. Dis.)
Kemp, T. A., 1092 (Haem.)
Kendall, D., with Collard, 903 (Am.)
Keneford, J. R. & Simpson, J. O. E., (1037) (Mal.)
Kennedy, J. S., 561 (Mal.)
Kerbrat, G., with Bénard, (514) (Typh.)
Kermack, W. O., with Hutchison, (877) (Mal.)
Kerr, R. W. & Rafferty, W. J., 624 (Ent.)
Kershaw, G. R., 749 (Misc. Dis.)
Kervran, P., 190 (Leish.)
—— & Aretas, R., 1018 (Der.)
Kerwin, J. F., with Campbell (34) (Mal.)
——, with ——, La Forge & Campbell, B. K., (34) (Mal.)
——, with —— & Helbing, (34) (Mal.)
——, with ——, Sommers & Campbell, S. K., (34) (Mal.)

Kessel, J. F. & Moore, F. J. with Kaplan, F. M. & Schireson, R., 524 (Am.)
Kessler, A., 130 (Misc. Dis.)
Kevorkova, V. I., 671 (Hel.)
Khalek, F. A., with Halawani, Nor-el-Din & Shaker, 99 (Hel.)
Khalil, M. B. & Hilmy, I. S., 97 (Hel.)
Khan, F., 234 (Vms.)
Khelevin, N. V., 680 (Ent.)
Khizanishvili, A. O., with Kamalov, Gordadze, Tsutsunava, Kamalova & Buguianishvili, 224 (Hel.)
Khoury, C., with Girardi, 125 (Der.)
Kiebl, E. & Siedek, H., 960 (Mal.)
Kierland, R. R., 499 (Mal.)
Kieser, J. A., 218 (Hel.)
Kiker, C. C., with Watson & Hess, 801 (Mal.)
Kikuth, W., with Schilling, I., 199 (Typh.)
King, E. J., 618 (Heat Str.)
King, F. E., with Acheson & Spensley, 964 (Mal.)
King, J. D., 282 (Mal.)
——, with Maegraith, Tottey, Adams & Andrews, 501 (Mal.)
King, W. V. & Hoogstraal, H., 543 (Ent.)
——, with Toffaleti, 958 (Mal.)
Kingery, R. M., with Bounds (429) (R. F.)
Kirby, F. G., 1060 (Typh.)
Kirk, J. B., 350 (Heat Str.)
Kirk, R., 986 (Leish.)
—— & Corkill, N. L., 122 (Vms.)
—— & Sati, M. H., 885 (Leish.)
Kitchen, S. F., with Boyd, 560 (Mal.)
—— & Putnam, P., 23 (Mal.)
Klein, I. F., with Greenberg, Pellitteri & Huebner, 707 (Typh.)
Kligler, I. J., with Birnbaum & Goldblum, 22 (Mal.)
Klinefelter, H. F., Jr., with Dell, 171 (Mal.)
Klyueva, N. G. & Roskin, G., (406) (Tryp.)
Knickerbocker, T. W., with Rakofsky, 236 (Der.)
Knies, P. T., with Gordon, (659) (Pl.)
Knipe, F. W., with Soper, Casini, Riehl & Rubino, 877 (Mal.)
Knipling, E. F., 135 (Ent.), (181) (Mal.)
——, with Madden & Linquist, 466 (Ent.)
——, with ——, Schroeder & Linquist, 544 (Ent.)
Knoll, E. W. & Howell, K. M., 524 (Am.)
Knowles, F. L., 700 (Mal.)
—— & Smith, C. S., 399 (Mal.)
Kodicek, E., Carpenter, K. J. & Harris, L. J., 119 (Def. Dis.)
Koenigsfeld, E. G. H. & Nambiar, K. P. S., (659) (Pl.)
Koepfli, J. B., Mead, J. F. & Brockman, J. A., Jr., (964) (Mal.)
Koepke, J. A., with Cole, 468 (Ent.)
Koerber, R., with Durieux & Boiron, 991 (Y.F.)
Kojevnikov, P. V., Dobrotvorsksya, N. V. & Latyshev, N. I., 940 (B.R.)
Kolodny, M. H., with Denhoff, 748 (Ulc.)
Koppisch, E., with Pomales Lebron, Arbona & Morales Otero, 198 (Typh.)
Koprowski, H., 580 (Y.F.)
——, with de Boer, Kunz & Cox, 712 (Den.)
—— & Cox, H. R., 518 (Den.)
Koransky, A., with Benedek, Lewe, Price & van Horn, 458 (Der.)
Kotby, A., 614 (Oph.)
Kouri, P., Iriondo, M. & Fusté, R., (903) (Am.)
——, with Milanes, Curbelo, Rodriguez & Spies, 452 (Sp.)
Koussitasseff, 638 (Mal.)
Kraan, H., with Swellengrebel, 286 (Mal.)
Kreisberger, J., with Passouant, (638) (Rab.)
Kremer, C. B., with Elderfield, Kupchan, Birstein & Cortes, (964) (Mal.)
Krichevski, A. M., 675 (Der.)
Krivonosova, Z. F., 161 (Mal.)
Krontovskaja, M. K., with Tovarnickij & Ceburkina, (301) (Typh.)
Kuhlenbeck, H., with Sadusk, 196 (Typh.)
Kuhn, H. S., 617 (Oph.)
Kuitunen-Ekbaum, E. & Webster, D., 671 (Hel.)
Kuntz, R. E., 330 (Hel.)
—— & Stirewalt, M. A., 434 (Hel.)
Kunz, L. J., with de Boer, Koprowski & Cox, 712 (Den.)
Kuo, S. C., 331 (Hel.)
Kupchan, S. M., with Elderfield, Kremer, Birstein & Cortes, (964) (Mal.)
Kuzenok, A., with Olitzki & Czaczkes, 195 (Typh.)
Kyker, G. C. & McEwen, M. M., 757 (Lab.)
——, —— & Cornatzer, W. E., 802 (Mal.)

L.

Lachnit, V., 704 (Typh.)
Ladell, W. S. S., 1097 (Heat Str.)
Laemmert, H. W., Jr., Ferreira, L. de C. & Taylor, R. M., 414 (Y.F.)
——, with Fox, 1062 (Y.F.)
van Laere, J., 394 (Mal.)
Laffer, N.C., with Sashin, Brown, McDowell & Boeing, 460 (Der.)
Lafferty, L. C., with Schnitzer & Buck, 185 (Tryp.)
La Forge, R. A., with Campbell, K. N., Kerwin & Campbell, B. K., (34) (Mal.)
Lagodsky, H., with Launoy, 702 (Tryp.)
Laha, P. N., 585 (Am.)
Lahiri, D. C., with Hazra & Sokhey, (235) (Vms.)
Laigret, J., 580 (Y.F.)
Laird, R. L., with Huff, Coulston & Porter, 1046 (Mal.)
de Lajudie, P., with Floch, 322, 589, 724, (727) (Lep.), 922 *bis* (Hel.), 1007 (Ys.), (1054) (Tryp.)
——, with —— & Taillefer-Grimaldi, 93 (R.F.)
Lal, R. B. & Seal, S. C., 144 (Reports, etc.)
Lal, S. B., with Basu, (860) (Reports, etc.)
Lalich, J. J., 1047 (Bl.)
Lam, F. K. & Price, S., 933 (Der.)
Lambillon, J., 1034 (Mal.)
Landau, H. D., (463) (Misc. Dis.)
Landau, J., with Feigenbaum, (618) (Oph.)
Landquist, J. K., with Curd & Rose, (699) (Mal.)
——, with Rose & Curd, (964) (Mal)
Landsborough, D. & Tunnell, N., 418 (Pl.)

Lane, J., with Cerqueira, 1063 (Y.F.)
Lang, W. R., 231 (Def. Dis.)
Lange, H. F., 547 (Lab.)
de Langen, C. D., 671 (Def. Dis.)
Langridge, W. P., with Lewis, 980 (Tryp.)
Languillon, J., 596 (Lep.)
Lanza, G., 160, 168 (Mal.)
Lapenta, R. G., with Ingraham, 212 (R.F.)
Lapeyssonnie, J. & Lapeyssonnie, L., 295 (Tryp.)
Laranja, F. S., with Dias & Nobrega, (651) (Tryp.)
Larmande, A., with Toulant, 1095 (Oph.)
Larsh, J. E., Jr., 222 (668) (Hel.)
Lassman, P., with Rodhain, 184 (Mal.)
Latif, N., (885) (Tryp.)
Latyshev, N. I., with Kojevnikov & Dobrotvorskaya 940 (B.R.)
Launoy, L. & Lagodsky, H., 702 (Tryp.)
—— & Prieur, M., Mlle., 509 (Tryp.)
Laurans, R., 468 (Ent.)
Laurie, E. M. O., 76 (Pl.)
Lauterburg-Bonjour, M., 310 (Y.F.)
Lavier, G., (813) (Y.F.)
Lavietes, P. H., with Most, Kane, London, Schroeder & Haymen, 496 (Mal.)
Laviron, P., with Beaudiment & Aretas, 727 (Lep.)
Lawrence, D., 209 (Am.)
Lawrence, J., with Coghill & Ballantine, 718 (R.F.)
Laws, S. G., 804 (Tryp.)
Lazarus, A. S. & Gunnison, J. B., 1064 (Pl.)
Leavis, H. (342) (Haem.)
Leavitt, S. S. & Beck, O. H., 916 (Hel.)
Lebied, B., with Wanson, 131 (Ent.)
Lebon, J., Choussat, H. & Duboucher, G., (525) (R.F.)
—— & Fabregoule, M., (665) (R.F.)
Le Bourdelles, B., with Swyngedauw & Masse, (789) (Mal.)
Lee, D. H. K., 1096 (Heat Str.)
——, with Macpherson & Furlonger, 934 (Heat Str.)
Lee, D. J., with Woodhill, 275 *bis* (Mal.)
Leech, R. B., 527 (Ys.)
Lees, A. D., 212 (R.F.)
Leeson, F., (458) (Vms.)
Lehmann, H., with Walters & Rossiter, 672 *bis* (Def. Dis.)
Lehmensick, R., 230 (Hel.)
Leitman, M. Z., with Sofiev, 906 (R.F.)
Lemierre, 192 (Typh.)
Lennox, B., with de Wardener, 450 (Def. Dis.)
Leonardon, H., with Benhamou, Destaing & Vargues, (586) (R.F.)
Leslie, A., with Silverman, 819 (Am.)
Lesses, M. F., with Finland, 1061 (Typh.)
Levaditi, C. & Vaisman, A., 186 (Tryp.), 718 (R.F.)
——, —— & Noury, H., 718 (R.F.)
Levick, C. B. & MacGregor, M. E., 789 (Mal.)
Levine, A. S., with Bianco, Saunders & Cohn, 1034 (Mal.)
Levine, H. D., 59 (Typh.)
—— & Erlanger, H., 395 (Mal.)
Levine, N. D. & Harper, P. A., 953 (Mal.)
Levinson, L. B. & Skadovskaja, N. S. 888 (Leish.)
Levy, M. D., with Arnold, 237 (Der.)
Lewe, I., with Benedek, Price, Koransky & van Horn, 458 (Der.)
Lewis, C. F. & Musselman, M. M., 531 (Def. Dis.)
Lewis, D. J., 656 (Y.F.)
Lewis, E. A. & Langridge, W. P., 980 (Tryp.)
Lewis, M. N., 406 (Tryp.)
Lewis, R., with Faust, Heilbrunn & Murray, 1067 (Am.)
Lewis, R. A., 317 (Am.)
Lewis's Medical, Scientific & Technical Library, 941 (B.R.)
Lewthwaite, R., O'Connor, J. L. & Williams, S. E., 410 (Typh.)
Liebow, A. A., MacLean, P. D., Bumstead, J. H. & Welt, L. G., 351 (Ulc.)
Lie Kian Joe, with Bonne & Bras, 1013 (Hel.)
Lieou, Y. C., with Raynal, 637 (Rab.)
Lifschitz, J., Umana, A. C. & Vergara, J. J., 276 (Mal.)
Lillie, R. D., 938 (Lab.)
Lincicum, D. R., with Martin, Graziani & Collins, 334 (Hel.)
Lindberg, K., 925 *bis* (Hel.)
Lindquist, A. W., with Cristol & Haller, 466 (Ent.)
——, with Madden & Knipling, 466 (Ent.)
——, with —— & Schroeder, 1042 (Mal.)
——, with —— & Knipling, 544 (Ent.)
Lindsay, J. K., 618 (Heat Str.)
——, with Caplan, 618 (Heat Str.)
Linduska, J. P., Cochran, J. H. & Morton, F. A., 681 (Ent.)
Lippincott, S. W., with Christianson, Gordon & Daniels, 24 (Mal.)
——, with Gordon & Christianson, 176 (Mal.)
——, with ——, Marble, Hesselbrook & Ellerbrook, 173 (Mal.)
——, Marble, A., Ellerbrook, L. D., Hesselbrock, W. B., Engstrom, W. W. & Gordon, H. H., 166 (Mal.)
——, Paddock, F. K., Rhees, M. C., Hesselbrock, W. B. & Ellerbrook, L. D., 601 (Hel.)
Lips, M., with Vincke & Cambier, 286 (Mal.)
Lisansky, E. T., with Downs & Harper, 953 (Mal.)
——, with Harper & Sasse, 953 (Mal.)
Lisitsa, F. M., 171 (Mal.)
Lisova, A. I., Sofiyev, M. S. & Eskin, V. A., 876 (Mal.)
Little, S. C., with Watson & Murphey, 916 (Hel.)
Liu, P. Y., with Reimann, Chang, Chu & Ou, 423 (Chl.)
Liu, W. T., 574 (Typh.)
Livadas, G. A. & Sphangos, J. C., 259 (B.R.)
Livesay, H. R., with Pollard, Wilson & Woodland, 574 (Typh.)
——, Wilson, D. J., Pollard, M. & Woodland, J. C., 206 (Den.)
Lizondo, J. E., with Romaña & Conejos, 300 (Mal.)
Lleras Restrepo, F., with Ignacio Chala, 823 (Lep.)
Llopart, J., with Chiffiet, (835) (Hel.)
Lôbo, R., 914 (Hel.)
Loeb, R. F., 396 (Mal.)

van Loghem, J. J., (421) (Chl.), (630) (Reports, etc.)
London, I. M., with Most, Kane, Lavietes, Schroeder & Haymen, 496 (Mal.)
Lopes, D. M., 328 (Hel.)
Lopez, G. G., Spies, T. D., Menendez, J. A. & Toca, R. L., 456 (Sp.)
——, with ——, Milanes & Aramburu, 845 (Sp.)
Lopez-Chavez, G. J. (337) (Hel.)
Losee, K., with Bernstein, Pribyl & Lott, (964) (Mal.)
Lott, W. A., with Bernstein, Pribyl & Losee, (964) (Mal.)
——, with ——, Stearns & Dexter, (964) (Mal.)
——, with ——, —— & Shaw, (964) (Mal.)
Loughlin, E. H. & Stoll, N. R., 335, 735 (Hel.)
——, with ——, Harris & Chenoweth, 444 (Hel.)
de Louveiro, J. A., with Monteiro, Coutinho & Janz, 240 (Def. Dis.)
Lourie, E. M., 1048 (Tryp.)
——, with Fulton, 403 (Tryp.)
——, with Williamson, 48 (Tryp.)
——, with —— & Bertram, 970 (Mal.)
Loutit, J. F., 743 (Haem.)
Louveaux, J., 1060 (Typh.)
Lovell, B. J., with Hull, Openshaw & Todd, (642) (Mal.)
Lovett, W. C. D., 1102 (Ent.)
Lowe, C. U. & Augustine, D. L., (754) (Misc. Dis.)
Lubitz, J. M., with Ecker, (986) (Leish.)
Lucké, B., 1048 (Bl.)
Luckey, T. D., Moore, P. R., Elvehjem, C. A. & Hart, E. B., 119 (Def. Dis.)
Ludwig, R. G. & Nicholson, H. P., 574 (Typh.)
Luis Carri, E., 663 (Am.)
Lum, L. C., 500 (Mal.)
Lupasco, G., with Cantacuzène, 529 (Hel.)
——, with Zotta, Radacovici, Cantacuzène & Teodoresco, 623 (Misc. Dis.)
Luppova, N. N., 639 (Mal.)
Lurye, A., 558 (Mal.)
Lutterloh, C. H. & Shallenberger, P. L., 177 (Mal.)
Luttermoser, G. W., 1078 (Hel.)
Lwoff, M., with Nicolle, (885) (Tryp.)

M.

McAllister, J., with Weinman, 1100 (Prot.)
McArthur, J., 788, 795 (Mal.)
McCall, J. V., Jr., with Page, 396 (Mal.)
McCance, R. A. & Black, D. A. K., 44 (Bl.).
Macchiavello, A., 78, 519, 997 (Pl.)
——, Mostajo, B. & Mostajo, B., Jr., 713 (Pl.)
McClymont, A. G., with Wu, Ghosh & Roy, 464 (Ent.)
McCollough, G. T., with Jones & Morton, 467 (Ent.)
McCulloch, R. N., 1020 (Ent.)
McDaniel, G. E., with Sabrosky & Reider, 392 (Mal.)
McDermott, E. B., with Wheeler, Adler & Khan, (642) (Mal.)
Macdonald, G., 182 (Mal.)
McDonald, S., 592 (Lep.)
McDowell, F. & Varney, P. L., 851 (Misc. Dis.)
McDowell, H. C., with Sashin, Brown, Laffer & Boeing, 460 (Der.)
McEwen, M. M., with Kyker, 757 (Lab.)
——, with —— & Cornatzer, 802 (Mal.)
McFadzean, A. J. S., 739 (Def. Dis.)
Macfarlane, D. G. & Macy, R. W., 97 (Hel.)
Macfarlane, J. O., with Groupe & Nigg, 197 (Typh.)
McGann, V. G., with Burrows, Mather & Wagner, 422 (Chl.)
McGee, H. R., with Rostorfer, 567 (Mal.)
MacGregor, M. E., with Levick, 789 (Mal.)
MacGregor, R. B., 360 (Misc. Dis.)
Machella, T. E., with Fine, R. & Burgoon, D. F., 557 (Mal.)
MacInnes, D. G., 701 (Mal.)
McIntyre, J. T., 618 (Heat Str.)
Mackay, A. G., 720 (Lep.)
McKee, R. W., with Anfinsen, Geiman, Ormsbee & Ball, 1043 (Mal.)
—— & Geiman, Q. M., 649 (Mal.)
——, with ——, Anfinsen, Ormsbee & Ball, 1043 (Mal.)
——, Ormsbee, R. A., Anfinsen, C. B., Geiman, Q. M. & Ball, E. G., 1043 (Mal.)
McKendrick, A. J., with Young & Farr, (426) (R.F.), (448) (Hel.)
Mackerras, I. M., 518 (Den.), 642, 874 (Mal.)
—— & Aberdeen, J. E. C., 555 (Mal.)
McKinney, W. L., Jr., with Monat, 90 (Am.)
MacLean, with Liebow, Bumstead & Welt, 351 (Ulc.)
McLennan, E. I., with Turner, Rogers & Matthaei, 371 (Lab.)
Macleod, J. M. H. & Muende, I., 255 (B.R.)
McLetchie, J. L., 971 *ter* (Tryp.)
McLimans, W. F. & Grant, C. W., 706 (Typh.)
——, with Tullis, Gersh, Jenney & Vinson, 895 (Typh.)
McMahon, J. P., 626 (Ent.)
——, with Garnham, 1084 (Hel.)
McMahon, M. C., with Wayson & Prince, 419 (Pl.)
McMartin, R. B., with Dimson, 32 (Mal.)
McMullen, D. B., 917 (Hel.)
—— & Graham, O. H., 917 (Hel.)
—— & Ingalls, J. W., 917 (Hel.)
——, with Wright, Faust & Bauman, 832 (Hel.)
Macpherson, A., with Hill & Findlay, 94 (Ys.)
Macpherson, K., 686 (B.R.)
Macpherson, R. K., Lee, D. H. K. & Furlonger, E., 934 (Heat Str.)
McRoberts, G. R., 1026 (B.R.)
Macy, R. W., with Macfarlane, 97 (Hel.)
Madden, A. H., Lindquist, A. W. & Knipling, E. F., 466 (Ent.)
——, Schroeder, H. O., Knipling, E. F. & Lindquist, A. W., 544 (Ent.)
——, — & Lindquist, A. W., 1042 (Mal.)
Maddux, H., with Richardson, Hewitt, Seager, Brooke & Martin, 289 (Mal.)
Madsen, H., 110, 840 (Hel.)
Maegraith, B., 121 (Sp.), 393 (Mal.)
——, Brundrett, J. C., Rigby, J. D. & Sladden, R. A., 886 (Leish.)
—— & Havard, R. E., 44 (Bl.)

Maegraith, B. G., *et al.*, 27, 28, 31, 498 *bis*, 499 *bis* (Mal.)
——, with Findlay, Markson & Holden, 501 (Mal.)
——, Tottey, M. M., Adams, A. R. D., Andrews, W. H. H. & King, J. D. 501 (Mal.)
Maffrand, R., 213 (Lep.)
Magrou, E. 417 (Pl.)
Mahaffy, A. F., with Haddow, Smithburn & Bugher, 992 (Y.F.)
——, Smithburn, K. C. & Hughes, T. P., 72 (Y.F.)
Maier, J., with Bang, Hairston & Trager, 281, 503, 696 (Mal.)
——, with Winkenwerder, Hunninen, Harrison, Billings & Carroll, 834 (Hel.)
Maisel, A. L., with Tallant & Rakestraw, 82 (Am.)
Maitra, S. R., with Sarkar, (846) (Vms.)
——, with —— & Roy, (846) (Vms.)
Majumder, D. N. & Das Gupta, C. R., 183 (Mal.)
Makar, N., 1010 (Hel.)
—— & Fawzy, R. M., 1011 (Hel.)
Makari, J. G., 279 (Mal), 298 (Leish.)
Makower, H., 1035 (Mal.)
Malamos, B., (188) (Leish.)
Malamud, N., Haymaker, W. & Custer, R. P., 1097 (Heat Str.)
Malanga, C., with Seeler, (399) (Mal.)
Malcolmson, M. E. & Wishart, F. O., 407, 408 (Typh.)
——, with —— 194 (Typh.)
Maldonado, J. F., (586) (Am.)
——, with Hernandez Morales & Pratt, 330 *bis* (Hel.)
Malone, R. H., 511 (Leish.)
Manceaux, A., Bardenat, C. & Susini, R., 849 (Misc. Dis.)
—— & Susini, R. (239) (Misc. Dis.)
Mandoul, R. & Pautrizel, R., 319 (Am.)
Manifold, J. A., 683 (Reports, etc.)
Maniscalco, G., 627 (Lab.)
Mankad, K. K., with Jhatakia, (663) (Am.)
Mann, F. G., with Chapman & Gibson, (1037) (Mal.)
——, Naylor, F. T. & Porter, J. W. G., (1037) (Mal.)
—— & Porter, J. W. G., (1037) (Mal.)
Mann, I., 1094 (Oph.)
Manson-Bahr, P., 360 (Misc. Dis.)
—— & Clarke, O., 456 (Sp.)
Manuwa, S. L. A., 626 (Ent.)
Manwell, R. D., 43, (802) (Mal.)
——, with Russell & West, 372 (B.R.)
Maple, J. D., with Hurlbut, Wilson, Fallander & Husman, 881 (Mal.)
——, with Travis, Hurlbut & Husman, 646 (Mal.)
Maqsood, M., (735) (Hel.)
Mara, L., with Jannone & Ferro-Luzzi, 251 (Reports, etc.)
Marano, A. & Niño, F., (1093) (Der.)
Marble, A., with Engstrom, Gordon & Brunsting, 961 (Mal.)
——, with Gordon, Dieuaide, Christianson & Dahl, 963 (Mal.)
——, with ——, Lippincott, Hesselbrock & Ellerbrook, 173 (Mal.)
Marble, A., with Lippincott, Ellerbrook, Hesselbrock, Engstrom & Gordon, 166 (Mal.)
Marcotorchino, M., (432) (Hel.)
Maren, T. H., with Otto, 1083 (Hel.)
Maria Guerrero, J., 718 (R.F.)
Mariano, J., with Costa, (726) (Lep.)
Marikovski, P. I., 503 (Mal.)
Markell, E. K., Mullinger, P. E. & Schneider, D. J., 597 (Hel.)
Markham, F. S., with Soper, Davis & Riehl, 892 (Typh.)
Marks, J. L., with Feinstein & Yesner, 66 (Typh.)
Markson, J. L., with Findlay, 884 (Bl.)
——, with ——, Maegraith & Holden, 501 (Mal.)
Maroun, T., 1035 (Mal.)
Marple, C. D., (986) (Leish.)
Marriott, H. L., 360 (Misc. Dis.)
Marrugat, O. L., (1082) (Hel.)
Marsden, A. T. H., 86 (Am.), 353 (Misc. Dis.)
Marshall, E. K., Jr. & Dearborn, E. H., 292 *bis* (Mal.)
——, with ——, 403, 568 (Mal.)
Martin, A. P., 140 (Reports, etc.)
Martin, C., with Fontan, Verger & Mouliès, 638 (Mal.)
Martin, F., with Richardson, Hewitt, Seager, Brooke & Maddux, 289 (Mal.)
Martin, L. A., with Blanc, (813) (Typh.)
——, with —— & Maurice, 898 (Typh.)
——, with Maurice & Blanc, 709 (Typh.)
Martin, W. B., Graziani, J. G., Collins, J. & Lincicum, D. R., 334 (Hel.)
Martinez, E. C., with Santiago-Stevenson & Hernandez-Morales, F., 902 (Am.)
Marucci, P. E., with Elmendorf, Griffin, Meyer & Ryan, 466 (Ent.)
Marvin, H. N., with Rigdon, 884 (Mal.)
Masillamani, S. G., (179) *bis*, 955 (Mal.)
Mason, P. K., Daniels, W. B., Paddock, F. K. & Gordon, H. H., 732 (Hel.)
Masse, L., with Swyngedauw & Le Bourdellès, (789) (Mal.)
Massoud, F., (618) (Oph.)
Mather, A. N., with Burrows, Elliott & Wagner, 422 (Chl.)
——, with ——, McGann & Wagner, 422 (Chl.)
Mather, K., 244 (Lab.)
Mathis, W. V., Ferguson, F. F. & Simmons, S. W., 644 (Mal.)
Matilla, V., Covaleda, J. & Aparicio Garrido, J., 279 (Mal.), 443 (Hel.) (608) (Haem.)
Matilla Gomez, V., 855 (Ent.)
Matthaei, E., with Turner, McLennan & Rogers, 371 (Lab.)
Mattingly, P. F., 365 (Ent.)
Maurice, A., with Blanc & Martin, 709, 898 (Typh.)
Mautner, L. S., with Ikejiani, 185 (Tryp.)
May, E. L., 729 (Hel.), (905) (Am.)
Mayer, M., with Ignacio Baldo, Gil Yepez & Pifano, 1077 (Hel.)
Mayer, M. M. & Heidelberger, M., 172 (Mal.)
Mazzotti, L., with Bozicevich, Donovan, Diaz & Padilla, 669 (Hel.)
Mead, J. F., with Koepfli & Brockman, (964 (Mal.)

Medina, H., with Pereira, 188 (Leish.)
Medulla, C., (598) (Hel.)
Megaw, J. W. D., 300 (Typh.)
de Meillon, B., 469 (Ent.)
—— & Golberg, L., 525 (R.F.)
Meira, J. A., (529), (825) (Hel.)
——, with de Oliveira, 668 (Hel.)
de Meira, M. T. V. & Coito, A. de M. F., 1014 (Hel.)
Meleney, H. E., 492 (Mal.)
—— & French, R. S., 410 (Typh.)
——, with Snyder, 582 (Am.)
Mellanby, K., 240, 364 (Ent.)
de Mendonça, F. C. & Cerqueira, N. L., 965 (Mal.)
Menendez, J. A., with Lopez, Spies & Toca, 456 (Sp.)
Menon, I. G. K., 852 (Misc. Dis.)
——, with Ahuja, Brooks & Veeraraghavan, (846) (Vms.)
Menon, K. P., with Cochrane & Pandit, 322 (Lep.)
Menyasz, E. & Nicolas, E., 892 (Typh.)
Mer, G., Birnbaum, D. & Aioub, A., 542 (Ent.)
Merchant, D. J., (806) (Tryp.)
Merchant, S. M., (457) (Haem.)
Mercier, C., with Roux, 207 (Pl.)
Merkel, W. C., 172 (Mal.)
Merskey, C., 1070 (R. F.)
Mesrobeanu, L., (349) (Der.)
Messini, M., (512) (Leish.)
Meulengracht, E., with Jersild, 457 (Haem.)
Meyer, E., with Baker, Sommer, Foster & Meyer, 712 (Pl.)
Meyer, H., 968 (Mal.)
Meyer, K. F., 816 (Pl.)
——, with Baker, Sommer, Foster & Meyer, 712 (Pl.)
Meyer, R., 195 (Typh.)
Meyer, S. L., with Elmendorf, Marucci, Griffin & Ryan, 466 (Ent.)
Meyers, M. C., with Bethell, Andrews, Swendseid, Bird & Brown, (533) (Haem.)
Mezey, C. M. & Fowler, R., (350) (Der.)
Michael, P., 210 (Am.)
Mider, G. B., Smith, F. D. & Bray, W. E., Jr., 610 (Der.)
Miescher, G., 347 (Der.)
Milanes, F., Curbelo, A., Rodriguez, A., Kouri, P. & Spies, T. D., 452 (Sp.)
——, with Spies, Lopez & Aramburu, 845 (Sp.)
Miller, J. F., with Einhorn, 215 (Hel.)
Mills, K. C., with van den Ende, Stuart-Harris, Fulton, Niven, Andrewes, Begg, Elford, White, Hawley, Hamilton & Thomas, 472 (B.R.)
Millspaugh, D. D. & Fuller, H. S., 705 (Typh.)
Milstone, J. H., with Jennison & Helwig, (679) (Misc. Dis.)
Ministry of Supply, (136) (Ent.)
Mirvish, I. & Hilson, D, 615 (Oph.)
Mitchell, J. G., with Heath, 754 (Ent.)
Miyara, S., Conte, D., Horenstein, B & Córica, P., 815 (Pl.)
Mofidi, C., with Baltazard & Bahmanyar, 905 (R.F.)
——, with ——, —— & Seydian, 906 (R.F.)
Mohamed, A. H., with Soliman, 458 (Vms.)
Mohapatra, G. S., (715) (Am.)
Moiser, B., 321, 725 (Lep.)
Molina, R. D. & Santos, H. A., (669) (Hel)
Molnar, G. W., Towbin, E. J., Gosselin, R. E., Brown, A. H. & Adolph, E. F., 461 (Heat Str.)
Moloshok, R. E., with Dack, 662 (Am.)
Mom, A. M., 594, 1074 (Lep.)
—— & Bernal, M., 596 (Lep.)
Monat, H. A. & McKinney, W. L., Jr., 90 (Am.)
Monod, T., 757 (Reports, etc.)
Monroe, L. & Hopper, J., Jr., 246 (Lab.)
Monteiro, A. M., Coutinho, H., Janz, G. J. & de Loureiro, J. A., 340 (Def. Dis.)
Montel, R. & Giroud, P., 322 (Lep.)
Montessori, P. P., 123 (Der.)
Montestruc, E., 641 (Mal.)
Montoya, J. A., (301) (Typh.)
Moore, F. J., with Kessel, Kaplan & Schireson, 524 (Am.)
Moore, P. R., with Luckey, Elvehjem & Hart, 119 (Def. Dis.)
Moorehead, M. T., 113 (Hel.)
Mooser, H., 191 (Typh.)
Morales Otero, P., with Pomales Lebron, Arbona & Koppisch, 198 (Typh.)
Morcos, F., with Halawani & Baz, 792 (Mal.)
Moreau, with Ouary, 353 (Ulc.)
Morehouse, W. G., with Burrows & Freed, 926 (Hel.)
Moreno Berdugo, J. & Infante Gómez, A., 820 (R.F.)
Moretti, G. F., with Bonnin & Dubois, 343 (Haem.)
Morgan, H. R., Stevens, D. A. & Snyder, J. C., 808 (Typh.)
Morgan, R. E., Sheppard, R. R. & Barnett, G. S., 1039 (Mal.)
Morkos, F., with Halawani & Baz, 698 (Mal.)
Morrill, A. W., Jr., with Dews, 134 (Ent.)
Morris, K. R. S., 51 (Tryp.)
Morris, T. L., with Hynes & Ishaq, 233 (Def. Dis.)
——, with ——, —— & Verma, 744 (Haem.)
Morrison, R. J. G. & Johnston, C. R. St., 742 (Sp.)
Mortimer, D. A., (165) (Mal.)
Morton, F. A., with Jones & McCullough, 467 (Ent.)
——, with Linduska & Cochran, 681 (Ent.)
Morton, T. C., 314 (Am.)
Moshkovski, S. D., 956 (Mal.)
Mosing, H., 1058 (Typh.)
Most, H., with Baroody, 333 (Hel.)
——, with Bartter, Cowie, Ness & Forbush, 1012 (Hel.)
——, with Eyles, 838 (Hel.)
——, Hayman, J. M., Jr. & Wilson, T. B., 443 (Hel.)
——, Kane, C. A., Lavietes, P. H., London, I. M. Schroeder, E. F. & Hayman, J. M., 496 (Mal.)
——, with Katzin, 440 (Hel.)
Mostajo, B., with Macchiavello & Mostajo, 713 (Pl.)
Mostajo, B., Jr., with Macchiavello & Mostajo, 713 (Pl.)
Moulder, J. W., (1052) (Tryp.)
Mouliès, A., with Fontan, Verger & Martin, 638 (Mal.)

Moutia, A., with Jepson & Courtois, 1030 (Mal.)
Mueller, M. G., with Carlson & Bissell, 39, 174 (Mal.)
Muende, I., with Macleod, 255 (B.R.)
Muesebeck, C. F. W., (364) (Ent.)
Muir, E., 327, 824, 1073 (Lep.)
Mukerji, A. K. & Bhaduri, N. V., 836 (Hel.)
——, —— & Narain, S., 599 (Hel.)
——, with Chaudhuri, 920 (Hel.)
Mukerji, B., with Bose & Iyengar, 189 (Leish.)
Mukhatcheva, O. N., 494 (Mal.)
Mukherjee, S. P., with Guha, (558) (Mal.)
Mukherji, N., with Dharmendra, (728) (Lep.)
Mullinger, P. E., with Markell & Schneider, 597 (Hel.)
Mulock Houwer, A. W., (1095) (Oph.)
Muniz, J. & de Freitas, G., 296 (Leish.), 571 (Tryp.)
Muñoz, N. T., (308) (Y.F.)
Muñoz Rivas, G., 1071 (Lep.)
Murgatroyd, F., 282 (Mal.) 360 (Misc. Dis.), 527 (Ys.)
Murphey, F., with Watson & Little, 916 (Hel.)
Murphy, J. N., with Irons & Wolfe, 710 (Typh.)
Murray, A. J., 1010 (Hel.)
Murray, M. L., with Faust, Heilbrunn & Lewis, 1067 (Am.)
Murray, N. L., with Gear, 702 (Typh.)
Murray, R. E., 476 (B.R.)
Murray-Lyon, R. M., (714) (Am.)
Musgrave, A. J., 58 (Typh.)
Musselman, M. M., with Lewis, 531 (Def. Dis.)

N.

Nagaty, H. F., (667) (Hel.)
Nájera Angulo, L., 179 *bis* (Mal.)
Nájera, L., 241 (Ent.)
Nambiar, K. P. S., with Koenigsfeld, (659) (Pl.)
Napier, L. E. 360 (Misc.), 790 (Mal.)
—— & Das Gupta, C. R., 1104 (B.R.)
Narain, S., with Mukerji & Bhaduri, 599 (Hel.)
Nardini, J. E., (674) (Def. Dis.)
Nargund, K. S., with Bhatt, (666) (Hel.)
——, with Patel, (666) (Hel.)
Nasr, M., (668) (Hel.)
Nassiruddin, M., with Rao, 16 (Mal.)
de Navaille, (586) (R.F.)
Naval Medical Research Institute, 330 (Hel.)
Naylor, F. G., with Mann & Porter, (1037) (Mal.)
Neal, R. A., (663) (Am.)
Negherbon, W. O., with Shih Lu Chang, 1056 (Leish.)
Neghme, A. & Faiguenbaum, J., 734 (Hel.)
Nelson, C. T., 705 (Typh.)
Nelson, E. C., Webb, J. E., Bayliss, M. & Starkey, G. S., 229 (Hel.)
Nelson, E. M., with Suárez, Welch, Heinle & Suárez, 607 (Sp.)
Ness, A. T., with Bartter, Cowie, Most & Forbush, 1012 (Hel.)
Nettel, F. R., (671) (Hel.)
Neujean, G., 56, 57, 58 (Typh.)
—— & Doucet, G., 57 (Typh.)
Neves, J. A. & Costa, O. G., 610 (Der.)
Newton, W. L. & Pratt, I., 337 (Hel.)
——, with ——, 104 (Hel.)
Ngu, D. V., with Galliard, (695) (Mal.), 733, (737) (Hel.)
Nguyen-Dinh-Diep, with Bruneau, 655 (Typh.)
Nicolas, E., with Menyasz, 892 (Typh.)
Nicolle, P. & Lwoff, M., (885) (Tryp.)
Nicholls, L., 258 (B.R.)
Nicholson, H. P., with Ludwig, 574 (Typh.)
Niemegeers, L., with Wittebolle, 427 (R.F.)
Nigeria, 971 *ter.* (Tryp.)
Nigg, C., with Groupe & Macfarlane, 197 (Typh.)
Niño, F., with Marano (1093) (Der.)
Niño, F. L., (125), 349 (Der.)
Nisbet, T. W., (877) (Mal.)
van Nitsen, R., 684 (B.R.)
Niven, J. S. F., with van den Ende, Stuart-Harris, Fulton, Andrewes, Begg, Elford, White, Hawley, Mills, Hamilton & Thomas, 472 (B.R.)
Nobrega, G., with Dias & Laranja, (651) (Tryp.)
Noehren, T. H., 23 (Mal.)
Noel, G., with Hentz, (406) (Leish.)
Nogueira, J. F. P., with Bastos da Luz, (934) (Der.)
Nolan, D. E., with Warshawsky & Abramson, (317) (Am.)
Nor-el-Din, G., with Halawani, Shaker & Khalek, 99 (Hel.)
Norman, A. P., 54 (Leish.)
Norviit, L., 653 *bis* (Typh.)
Nouchy, with Benhamou & Sebah, 238 (Misc. Dis.)
Noury, H., with Levaditi & Vaisman, 718 (R.F.)

O.

Ober, R. E., 448 (Hel.)
Oberg, M. W., with Jones & Titus, (467) (Ent.)
Oberlé, G., 159 (Mal.)
O'Brien, H. R., 250 (Reports, etc.)
O'Brien, T. P., (547) (Reports, etc.)
O'Connor, J. L., with Lewthwaite & Williams, 410 (Typh.)
O'Donnell, J. E., 340 (Def. Dis.)
Odum, E. P. & Sumerford, W. T., (467) (Ent.)
Oleesky, S., with Jackson, (321) (R.F.)
Olitzki, L. & Buechler, E., 196 (Typh.)
——, with Bernkopf & Stuczynski, 587 (R.F.)
——, Czaczkes, J. W. & Kuzenok, A., 195 (Typh.)
de Oliveira, A., 285 (Mal.)
de Oliveira, H. L. & Meira, J. A., 668 (Hel.)
de Oliveira, P. P., 96 (Hel.)
de Oliveira Castro, G. M., 1072 (Lep.)
Oliver-González, J., 226, 729 (Hel.), 529 (Mal.)
——, with Culbertson & Rose, 912 (Hel.)
——, with ——, ——, Hernández Morales & Pratt, 924 (Hel.)
—— & Hernández Morales, F. 830 (Hel.)
——, with ——, (229) (Hel.)
——, with —— & Pratt, 434, 731 (Hel.)
——, with ——, Suárez & Pratt, 218 (Hel.)
——, with Pratt, 826 (Hel.)

Olle, R. G. & Hector Cambiaso, R., 1095 (Oph.)
Olson, B. J., Bell, J. A. & Emmons, C. W., 848 (Der.)
Olson, T. A., (131) (Ent.)
——, with Davis & Pollard, 303 (Typh.)
Oman, P. W. & Christenson, L. D., 953 (Mal.)
Onsy Bey, A., 1011 (Hel.)
Openshaw, H. T., with Hull, Lovell & Todd, (642) (Mal.)
Oppel, T. W., with Weber & Raymond, 206 (Den.)
Oppenheim, E., with Willett, 460 (Der.)
Oppenheim, J. M., Whims, C. B. & Frisch, A. W., 449 (Hel.)
Orenstein, A. J., (71) (Y.F.)
de Oreo, G. A., (458) (Vms.)
Orgain, H., with Gartrell, 20 (Mal.)
Ormsbee, R. A., with Anfinsen, Geiman, McKee & Ball, 1043 (Mal.)
——, with Geiman, Anfinsen, McKee & Ball, 1043 (Mal.)
——, with McKee, Anfinsen, Geiman & Ball, 1043 (Mal.)
Orozco, G., with Ruiz Sanchez & Ponce de Leon, 304 (Typh.)
Osburn, L. W., (513) (Typh.)
Osorno-Mesa, E., with Anderson, 309 (Y.F.)
Ostroumov, V. G., 904 (Am.)
Osvaldo Curci, A., with Federico Guillot, 591 (Lep.)
Oteiza Setien, A., Ibarra Perez, R. & Gonzalez Prendes, M. A., 95, 589 (Lep.)
Ott, W. H., with Seeler, 41, 185 (Mal.)
Otto, G. F., with Donaldson (335) (Hel.)
—— & Maren, T. H., 1083 (Hel.)
Ou, Y., with Reimann, Chang, Chu & Liu, 423 (Chl.)
Ouary & Moreau, 353 (Ulc.)
Oz, T. V., 59 (Typh.)

P.

Packalén, T., 809 (Typh.)
Packchanian, A. & Price, T. G., 429 (R.F.)
—— & Sweets, H. H., Jr., (651) (Tryp.)
Paddock, F. K., with Lippincott, Rhees, Hesselbrock & Ellerbrook, 601 (Hel.)
——, with Mason, Daniels & Gordon, 732 (Hel.)
Padilla, B. E., 192 (Typh.)
Padilla, E., with Bozicevich, Donovan, Mazzotti & Diaz, 669 (Hel.)
Padron Ibarbia, F., (715) (Am.)
Page, F. T., 424 (Am.)
Page, I. H., with Corcoran, 539 (Misc. Dis.)
Page, S. G., Jr. & McCall, J. V., Jr., 396 (Mal.)
Palmer, E. D., 731 (Hel.)
——, with Hollands, 439 (Hel.)
Pampana, (274) (Mal.)
Pandit, C. G., with Cochrane & Menon, 322 (Lep.)
Panja, D., Dey, N. C. & Ghosh, L. M., 1018 (Der.)
Panja, G., 214 (Lep.)
—— & Das, N. N., 999 (Chl.)
—— & Ghosh, S. K., 998 (Chl.)
Paquier, P., with Ceccaldi, Trinquier, Pelissier and Vargues, 949 (Rab.)
Pará, M., 628 (Lab.)
Pardo-Castello, V., Tiant, F. R. & Piñeyro, R., 1074 (Lep.)
Parikh, R. O., with Viswanathan, 798 (Mal.)
Parrinello, G., 297 (Leish.)
Parrot, L. Catanei, A. & Collignon, E., 700 (Mal.)
—— & Durand-Delacre, R., (754) (Ent.)
—— & Habibi, A., (132) (Ent.)
—— & Wanson, L., (132) (Ent.
Parvis, D., 1002 (Am.)
Pasfield, G., with Woodhill, 365 (Ent.)
Pasquet, P. & Haudon, J., 127 (Heat Str.)
Passouant, P. & Kreisberger, J., (638) (Rab.)
Patel, J. C., (609) (Haem.)
Patel, N. Z. & Nargund, K. S., (666) (Hel.)
Patiño Camargo, L., Cortés, E., García, V., Santos, J., Zozaya, C., Rueda, V. & Rosa del Valle, J., 1037 (Mal.)
Paton, J. P. J., Peck, C. R. & van de Schaaf, A., 622 (Misc. Dis.)
Patterson, H. M., 1007 (R.F.)
de Paula, A., 201 (Typh.)
Paulley, J. W. & Aitken, G. J., 120 (Sp.)
Pautrizel, R., with Mandoul, 319 (Am.)
Pavlov, P., (1020) (Ent.)
Pavlovsky, E. N., 376 (B.R.)
Payne, E. H., Balthazar, E. & Bezerra, D. A., 502 (Mal.)
——, —— & Fernandes, J. S., 502 (Mal.)
—— & Sanches, A., 692 (Mal.)
Payne, G. C., with Gahan, 880 (Mal.)
Payzin, S., 703 (Typh.), 717 (Am.)
Peacock, W. C., with Ferrebee & Gibson, 17 (Mal.)
Pearce, E., with Culbertson, 229 (Hel.)
Pease, E. D., with Kark, Aiton, Bean, Henderson, Johnson & Richardson, 842 (Def. Dis.)
Peck, C. R., with Paton & van de Schaaf, 622 (Misc. Dis.)
—— & Zwanenburg, T., 622 (Misc. Dis.)
Peck, J. L., Jr., with Stoll & Chenoweth, 737 (Hel.)
Peel, E. & Chardome, M., 339 (Hel.)
——, with Henrard & Wanson, 103 (Hel.)
——, with van Hoof & Henrard, 979 (Tryp.)
—— & Rodhain, J., 967 (Mal.)
Pégullo, J., with Raynaud, 93, (93) (R.F.)
——, with —— & Aubin, (718) (R.F.)
Pelissier, A., with Ceccaldi, Paquier, Trinquier & Vargues, 949 (Rab.)
Péllissier, A., 975 (Tryp.)
Pellitteri, O., with Greenberg, Klein & Huebner, 707 (Typh.)
Pellitteri, O. J., with Greenberg & Jellison, 990 (Typh.)
Peltier, (1063) (Y.F.)
Peltier, M., 417 (Y.F.), (426) (R.F.)
Pendse, G. S., Gokhale, V. G., Phalnikar, N. L. & Bhide, V. B., (681) (Ent.)
Penfound, W. T., Hall, T. F. & Hess, A. D., 21 (Mal.)
Penn, G. H., (754) (Ent.)
Pereira, C. & Medina, H., 188 (Leish.)
Pereira, J. M., with Fonseca, Cambournac, Pinto & Cunha, 487 (Mal.)
Pereira, M. de C., 1019 *bis* (Ent.)
Perez-Santiago, E., with Diaz-Rivera & Santos, 199 (Typh.)

Perkins, E. S., with Quin, 92 (R.F.)
Perlingiero, J. G. & György, P., (622) (Misc. Dis.)
Perlowagora, A. & Hughes, T. P., 657 (Y.F.)
Perret-Gentil, A., 396 (Mal.)
Perry, J. S., 76 (Pl.)
Perry, W. J., 391 (Mal.)
Perves, M., 277 *bis* (Mal.)
Pesigan, T. P., 1013 (Hel.)
—— & Garcia, E. Y., 244 (Lab.)
——, Reyes, F. A., Jr. & Yogore, M.G., Jr., (684) (Reports, etc.)
Pessôa, S. B., 939 (B.R.)
—— & Barretto, M. P., 300 (Leish.)
Peterkin, G. A. G., (684) (Reports, etc.)
—— & Hair, H. C., 643 (Mal.)
Peters, L., with Bueding & Waite, 667 (Hel.)
——, with Welch, Bueding, Valk & Higashi, 1015 (Hel.)
Peterson, J., Arbona, G. & Acosta, J., (224) (Hel.)
Peterson, J. S., 1065 (Chl.)
Peterson, O. L. & Fox, J. P., 813 (Typh.)
Petrjaev, E. D., 515 (Typh.)
Peyri, A., 1075 (Lep.)
Phalnikar, N. L., with Pendse, Gokhale & Bhide, (681) (Ent.)
Phelps, A. J., with Findlay & Hardwicke, 406 (Tryp.)
Philip, C. B., 989, 990 (Typh.)
—— & Woodward, T. E., 305 (Typh.)
Philip, M. I., with Rao, 1031 (Mal.)
Phillips, H. A., 606 (Def. Dis.)
Pick, F., 582, 661 (Am.)
Piédrola Gil, G., 855 (Ent.)
—— & Benitez Calvo, L. A., (636) (Rab.)
—— & Diez Melchor, F., 636 (Rab.)
Pielou, D. P., 134 (Ent.)
Pieraerts, G., 225 (Hel.)
Pieris, M. V. P., with de Saram, 447 (Hel.)
Pierron, A., 885 (Tryp.)
Piers, F., 889 (Leish.), 1085 (Def. Dis.)
Pifano, C. F., with Anduze & Vogelsang, (680) (Ent.)
——, with Ignacio Baldo, Gil Yepez & Mayer, 1077 (Hel.)
Pijoan, M., with Jachowski, 368 (Ent.)
——, Gerjovich, H. J., Hopwood, M. L., Jachowski, L. A. & Romine, J. T., 369 (Ent.)
Pijper, A., 1090 (Haem.)
Pinedo, C., Cánepa, C. H. & Fukelman, R., 532 (Def. Dis.)
Piñeyro, R., with Pardo-Castello & Tiant, 1074 (Lep.)
Pinilla, B. C., 529 (Hel.)
Pinto, A. R., (1052) (Tryp.)
Pinto, C., 216 (Hel.)
—— & de Almeida, A. F., 96, 216 (Hel.)
Pinto, M. R., 812 (Typh.), 821 (R.F.)
——, with Fonseca, Cambournac, Pereira & Cunha, 487 (Mal.)
Pizzi, P. T., (511) (Tryp.)
Planet, N. G., Cuocolo, R. & de Almeida, W. F., 90 (Am.)
Platt, B. S., 683 (Reports, etc.)
Pletnev, E. A., 957 (Mal.)
Plotz, H., 201 (Typh.)
——, Smadel, J. E., Bennett, B. L., Reagan, R. L. & Snyder, M. J., 204 (Typh.)
Plummer, P. J. G., (460) (Der.)
Pogge, R. C., with Faget, (728) (Lep.)
——, with —— & Johansen, F. A., 326 *bis* (Lep.)
——, with ——, ——, Fite, Prejean & Gemar, 594 (Lep.)
—— & Ross, H., 593 (Lep.)
Polak, M. F., with Snijders & Hoekstra, 993 (Y.F.)
Poletti, J. G., with Ginés, 528 (Lep.)
Pollard, M., with Davis, 303 (Typh.)
——, —— & Olson, T. A., 303 (Typh.)
——, with Livesay, Wilson & Woodland, 206 (Den.)
——, Wilson, D. J., Livesay, H. R. & Woodland, J. C., 574 (Typh.)
Pomales Lebron, A., Arbona, G., Morales Otero, P. & Koppisch, E., 198 (Typh.)
Pomerantz, C., with Huebner & Jellison, 411 (Typh.)
Ponce Caballero, L., 54 (Tryp.)
Ponce de Leon, E., with Ruiz Sanchez & Orozco, 304 (Typh.)
Ponce Pinedo, A. M., 826 (Hel.)
Pond, H. S., with Sayen, Forrester & Wood, 305 (Typh.)
Pontecorvo, G., 245 (Lab.)
Pontes, J. P. L., 211 (Am.)
Popov, I. S., 348 (Der.)
Porter, C. C., with Hellerman & Bovarnick, 400 (Mal.)
Porter, J. W. G., with Mann, (1037) (Mal.)
——, with —— & Naylor, (1037) (Mil.)
Porter, R. J., with Huff, Coulston & Laird, 1046 (Mal).
Portwood, L. M. & Sanders, E. 448 (Hel.)
Pospelova-Strom, M. V. & Tiburskaya, N. A., 213 (R.F.)
Postmus, S., with van Veen, 1085 (Def. Dis.)
Potop, I. & Bals, M., 301 (Typh.)
Potts, S. F., (681) (Ent.)
Pratt, C. K., with Culbertson, Rose, Hernández Morales & Oliver González, 924 (Hel.)
——, with Hernández Morales & Oliver González, 731 (Hel.)
——, with ——, Suárez & Oliver González, 218, 434 (Hel.)
—— & Oliver González, J., 826 (Hel.)
Pratt, H. D., with Stephens, 564 (Mal.)
Pratt, I. & Newton, W. L., 104 (Hel.)
——, with ——, 337 (Hel.)
Pratt-Thomas, H. R. & Cannon, W. M., 363 (Prot.)
Prejean, B. M., with Faget, Pogge, Johansen, Fite & Gemar, 594 (Lep.)
Preobrashenski, A. A., 898 (Typh.)
Preuss, H., with von Bormann, 891 (Typh.)
Pribyl, E. J., with Bernstein, Losee & Lott, (664) (Mal.)
Price, H., with Benedek, Lewe, Koransky & van Horn, 458 (Der.)
Price, S., with Lam, 933 (Der.)
Price, T. G., with Packchanian, 429 (R.F.)
Prieur, M., with Launoy, 509 (Tryp.)
Prince, F. M. & Wayson, N. E., 816, 1064 (Pl.)
——, with —— & McMahon, 419 (Pl.)
Pringle, G. & Singh, H., 853 (Ent.)
Prior, I. A. M., (836) (Hel.)
Proc. Roy. Soc. Med., 743 (Haem.)
Proske, H. O., with Brooke, 22 (Mal.)
Provost, C. & Flandrin, J., (1006) (R. F.)

Pryor, T., 618 (Heat Str.)
Psenichnov, A. V. & Raikher, B. I., 655 (Typh.)
Public Health Rep., (712) (Pl.)
Pullen, W., 790 (Mal.)
Putnam, P. & Hackett, L. W., 15 (Mal.)
——, with Kitchen, 23 (Mal.)

Q.

Queensland Institute of Medical Research, 757 (Reports, etc.)
Queiroz, E. J. S., with Cambournac & Simões, 181 (Mal.)
Quérangal des Essarts, J., Soubigou, X. & Autheman, (664) (Am.)
Quevauviller, A., (26), (792) (Mal.)
Quin, C. E. & Perkins, E. S., 92 (R.F.)
Qutubuddin, M., with Farooq, 669 (Hel.)

R.

Rabkin, R., with Wicht, 514 (Typh.)
Rachou, R. G., 165 *bis* (Mal.)
—— & Ferreira, M. O., 1032 (Mal.)
Radacovici, E., with Zotta, Cantacuzène, Lupasco & Teodoresco, 623 (Misc. Dis.)
Radna, R., 94 (Ys.), 99 (Hel.), 324 (Lep.)
Radovici, A., 363 (Misc. Dis.)
Rafferty, W. J., with Kerr, 624 (Ent.)
Rafyi, A., 92 (R.F.)
Ragan, C. A., with Robbins, 62 (Typh.)
Rai, B. B., 559 (Mal.)
Raikher, B. I., with Pshenichnov, 655 (Typh.)
Rail, G. A., 87, 522 (Am.)
Raison, C. G., with Curd, (699) (Mal.)
——, with —— & Rose, (1037) (Mal.)
Rajindar Pal, 19 (Mal.)
Rakestraw, M. R., with Tallant & Maisel, 82 (Am.)
Rakofsky, M. & Knickerbocker, T. W., 236 (Der.)
Ramanujain, K., with Cochrane, 323 (Lep.)
Rambert, P., with Bénard, Cajdos & Van den Broucke, 533 (Haem.)
Ramon, G., Girard, G. & Richou, R., 900 (Pl.)
Rangam, C. M., with Reddy, 583 (Am.)
Ransford, O. N., (129) (Misc. Dis.)
Rao, B. A., 16 (Mal.)
—— & Nassiruddin, M., 16 (Mal.)
Rao, H. R., with Rao, R. B. & Sundaresan, 952 (Mal.)
Rao, K. S. K., with Acharya, 681 (Ent.)
Rao, M. N. & Karmakar, G., 1089 (Haem.)
——, with Yaglou, 677 (Heat Str.)
Rao, R. B., Rao, H. R. & Sundaresan, B., 952 (Mal.)
Rao, S. R., 816 (Pl.)
Rao, T. R., with Viswanathan & Rao, T. S. R., 38 (Mal.)
Rao, T. S. R., with Viswanathan & Rao, T. R., 38 (Mal.)
Rao, V. V. & Philip, M. I., 1031 (Mal.)
——, with White, R. S., 491, 794 (Mal.)
——, with —— & Ghosh, 37 (Mal.)
Raoult, A., with Bergeret, (426) (R.F.)
Raper, A. B., with Kekwick & Wright, (674) (Def. Dis.)
Raskan, S. B., 606 (Def. Dis.)
Raubitschek, F., 849 (Der.)
Ravenel, S. F., 708 (Typh.)
Rawson, G. W. & Hitchcock, D. J., 716 (Am.)
——, with ——, 84 (Am.)
Raymond, R. W., with Weber & Oppel, 206 (Den.)
Raymond, W. D., 962 (Mal.)
Raynal, J. H., 893, 1059 (Typh.)
—— & Lieou, Y. C., 637 (Rab.)
Raynaud, R., Aubin, H. & Pegullo, J., (718) (R.F.)
—— & Pégullo, J., (93) (R.F.)
Read, H., with Thomen, 210 (Am.)
Read, H. S., Becker, F. T. & Kaplan, L. I., 177 (Mal.)
——, with ——, —— & Boyd, 25, 280 (Mal.)
——, with Glenn, Kaplan & Becker, 167 (Mal.)
——, with Kaplan, Becker & Boyd, 281 (Mal.)
Reagan, R. L., with Plotz, Smadel, Bennett & Snyder, 204 (Typh.)
Reardon, L. V., with von Brand, Rees & Simpson, 84 (Am.)
Redaelli, P., (125) (Der.)
Reddy, D. G. & Rangam, C. M., 583 (Am.)
Reed, H., 615 (Oph.)
Reed, J. G., 614 (Oph.)
Reenstierna, J., 728 (Lep.)
Rees, C. W., with von Brand, Reardon & Simpson, 84 (Am.)
Rees, W. H., with Henry, 676 (Heat Str.)
Regala, A. C., with Stransky, 343 (Haem.)
Reider, R. F., with Sabrosky & McDaniel, 392 (Mal.)
Reimann, H. A., Chang, G. C. T., Chu, L. W., Liu, P. Y. & Ou, Y., 423 (Chl.)
Rein, C. R. & Bossak, H. N., 136 (Lab.)
Reithmann, E., 653 (Typh.)
Remlinger, P. & Bailly, J., 949, 950, 1030 (Rab.)
Rennie, J. L., with Brown, 395, 497 (Mal.)
Reuter, A., with Schäfer, 1061 (Typh.)
Rey, M., with Bacigalupo & Toce, 529 (Hel.)
Reyes, F. A., Jr., with Pesigan & Yogore, (684) (Reports, etc.)
Rhees, M. C., with Lippincott, Paddock, Hesselbrock & Ellerbrook, 601 (Hel.)
Rhodesia, Southern, 1076 (Hel.)
Ribbands, C. R., 180, 795 (Mal.), 364 (Ent.), 463 (Misc.), 1041 (Mal.)
Rice, M. E., with Watson, 35 (Mal.)
Rice, R. I., Johnstone, H. F. & Kearns, C. W., 544 (Ent.)
Richardson, A. P., Hewitt, R. I., Seager, L. D., Brooke, M. M., Martin, F. & Maddux, H., 289 (Mal.)
Richardson, L. M., with Kark, Alton, Pease, Bean, Henderson & Johnson, 842 (Def. Dis.)
Richou, R., with Ramon & Girard, 900 (Pl.)
Riehl, L. A., with Soper, Davis & Markham, 892 (Typh.)
——, with ——, Knipe, Casini & Rubino, 877 (Mal.)
van Riel, J., 320 (R.F.), (561) (Mal.)
Rieman, E. H., 207 (Pl.)
Rietti, F., 745 (Haem.)
Rietz, J. H., 950 (Rab.)
Rifkin, H., with Zarrow, 328 (Hel.)
Rigby, G., with Brindle, 370 (Lab.)

Rigby, J. D., with Maegraith, Brundrett & Sladden, 886 (Leish.)
Rigdon, R. H., 884 (Mal.)
——, with Fletcher, 42 (Mal.)
—— & Marvin, H. N., 884 (Mal.)
—— & Rostorfer, H. H., (883), 883 (Mal.)
—— & Varnadoe, N. B., 505 (Mal.)
Rights, F. L., with Smadel, Jackson & Bennett, 513 (Typh.)
van Riper, W., 123 (Vms.)
Ripley, H. S., 200 (Typh.)
Ritchken, J. & Kantor, F., 1078 (Hel.)
Robbins, F. C., Gauld, R. L. & Warner, F. B., 63 (Typh.)
—— & Ragan, C. A., 62 (Typh.)
—— & Rustigian, R., 65 (Typh.)
——, ——, Snyder, M. J. & Smadel, J. E., 64 (Typh.)
Roberts, E. W., 1001 (Am.)
Robertson, A. G., with Wilson, 1039 (Mal.)
Robertson, E. G., 624 (Prot.)
Robertson, K., 428 (R.F.)
Robertson, K. M., 1068 (Am.)
Robertson, W. M. & Findlay, G. M., 747 (Haem.)
——, with —— & Zacharias, 344 (Haem.)
Robin, C. & Brochen, L., 643 (Mal.)
Robinson, R. A., (209) (Am.)
Robson, J. S., 930 (Sp.)
Roca-Garcia, M., with Anderson, 992 (Y.F.)
—— & Bates, M., 580 (Y.F.)
——, with ——, 308, 310, 415 (Y.F.)
Roche, with Sarrouy & Cabannes, 807 (Leish.)
Rockefeller Foundation, 247 (Reports, etc.)
Roden, A. T., with Etherington, Hickling & Sellick, 163 (Mal.)
Rodhain, J., 340 (Hel.), 801 (Mal.), (806) *bis*, (807) *bis* (Tryp.)
—— & Bone, G., (807) (Tryp.)
—— & Henry, E., (807) (Tryp.)
—— & Lassman, P., 184 (Mal.)
——, with Peel, 967 (Mal.)
Rodriguez, A., with Milanes, Curbelo, Kouri & Spies, 452 (Sp.)
Rodriguez-Molina, R. & Schwachman, H., 829 (Hel.)
Rogers, J. S., with Turner, McLennan & Matthaei, 371 (Lab.)
Rolda, H., with Calubaquib, 1100 (Prot.)
Romaña, C., Conejos, M. & Lizondo, J. E., (Leish.) 300
—— & Gil, J., 405 (Tryp.)
——, —— & de Romaña, M. S., 404 (Tryp.)
de Romaña, M. S., with Romaña & Gil, 404 (Tryp.)
Romine, J. T., with Pijoan, Gerjovich, Hopwood & Jachowski, 369 (Ent.)
Ronse, M., with Bruynoghe, 822 (R.F.)
Roos, A., Hegsted, D. M. & Stare, F. J., 403 (Mal.)
Roose, D. J., with Russell & Eisenhower, 425 (Am.)
Rosa del Valle, J., with Patiño Camargo, Cortés, García, Santos, Zozaya & Rueda, 1037 (Mal.)
Rose, F. L., with Curd, 283 (Mal.)
——, with ——, Davis & Hoggarth, (964) (Mal.)
——, with —— & Landquist, (964) (Mal.)
Rose, F. L., with Curd & Laudquist, (669) (Mal.)
——, with —— & Raison, (1037) (Mal.)
——, with Gage, 500 (Mal.)
Rose, H. M., with Culbertsan, Hernández Morales F., Oliver González & Pratt, 924 (Hel.)
——, with —— & Oliver-Gonzalez, 912 (Hel.)
——, with Gellhorn & Culbertson, 602 (Hel.)
Rose, J. R., 85 (Am.)
Rose, N. H., 353 (Misc. Dis.)
Rosenberger, H. G., with Smith, Beard & Whiting, 537, 675 (Der.)
Rosenstiel, (715) (Am.)
Rosenthal, S. R. & Routien, J. B., 460 (Der.)
Roskin, G., with Klyueva, (406) (Tryp.)
Ross, H., with Pogge, 593 (Lep.)
Ross Institute of Tropical Hygiene, India Branch, 547 (Reports, etc.)
Rossell, C. S. C., (1072) (Lep.)
Rossiter, R. J., with Walters & Lehmann, 672 *bis* (Def. Dis.)
Rostorfer, H. H. & McGee, H. R., 567 (Mal.)
——, with Rigdon, (883), 883 (Mal.)
Rotenburg, S. S., 27 (Mal.) 515 (Typh.)
Roth, H., 112 (Hel.)
Rousselot, R., (1063) (Pl.)
Routien, J. B., with Rosenthal, 460 (Der.)
Roux, A. H. & Mercier, C., 207 (Pl.)
Roy, D. N., with Wu, Ghosh & McClymont, 464 (Ent.)
Roy, P. K., with Sarkar & Maitra, (846) (Vms.)
Rubino, A., with Soper, Knipe, Casini & Riehl, 877 (Mal.)
Rueda, V., with Patiño Camargo, Cortés, Garcia, Santos, Zozaya & Rosa del Valle 1037 (Mal.)
Ruffin, J. M., with Darby & Cherrington, 456 (Sp.)
Ruiz Reyes, F., 1015 (Hel.)
Ruiz Sanchez, F., Ponce de Leon, E. & Orozco, G., 304 (Typh.)
Russell, B., (963) (Mal.)
Russell, B. A. S., 18 (Def. Dis.)
Russell, H. K., Eisenhower, E. W. & Roose, D. J., 425 (Am.)
Russell, P. F., West, L. S. & Manwell, R. D., 372 (B.R.)
Rustigian, R., with Robbins, 65 (Typh.)
——, with ——, Snyder & Smadel, 64 (Typh.)
Ryan, G. S., with Elmendorf, Marucci, Griffin & Meyer, 466 (Ent.)
Ryder, J. B. & Towson, R. T., 393 (Mal.)
Ryrie, G. A., 720 (Lep.)

S.

Sabalette, R., Dominguez, M. & Iglesias, R., 664 (R.F.)
Sabin, A. B. & Blumberg, R. W., 814 (Den.)
Sabrosky, C. W., 556 (Mal.)
——, McDaniel, G. E. & Reider, R. F., 39[illegible] (Mal.)
Sadovsky, A., with Adler & Bichovsky, (624) (Prot.)
Sadusk, J. F., Jr., 811 (Typh.)
—— & Kuhlenbeck, H., 196 (Typh.)
Saferstein, T. H., with Shaul, 665 (R.F.)
Sagher, F., 539 (Misc. Dis), 848 (Der.), 88[illegible] (Leish.)

Sagher, F., with Dostrovsky, (353) (Ulc.), 987, 1056, (Leish.)
Saglam, T., 610 (Der.)
Salazar Leite, A. & Bastos da Luz, J., 932, (934) (Der.)
Salem, H. H., with Attiah & El-Gammal, 618 (Oph.)
Salter, J. G., (937) (Misc. Dis.)
Saltzman, A., 988 (Leish.)
Sammon, G. K., with Fletcher & Dougan, (209) (Am.)
Samuel, E., with Drew & Dixon, 453 (Sp.)
Sanches, A., with Payne, 692 (Mal.)
Sanders, E., with Portwood, 448 (Hel.)
Sandor, G., with Girard, 900 (Pl.)
Sandoval, A. M., with Beltrán, 160 (Mal.)
Sandoval, C. A., with Beltrán, 961 (Mal.)
——, with —— & Dávalos, 968 (Mal.)
Sanjuan Fuentes, J., 486 (Mal.)
Santiago-Stevenson, D., Martinez, E. C. & Hernandez-Morales, F., 902 (Am.)
Santo, 654 (Typh.)
Santos, H. A., with Molina, (669) (Hel.)
Santos, J., with Patiño Camargo, Cortés, Garcia, Zozaya, Rueda & Rosa del Valle, 1037 (Mal.)
Santos, J. J., with Diaz-Rivera & Perez-Santiago, 199 (Typh.)
Santos Zetina, F., 240 (Prot.), (1019) (Der.)
Santra, I., with Dharmendra, 324 (Lep.)
Sapero, J. J., (274) (Mal.), 472 (Reports, etc.)
——, with Butler, 1034 (Mal.)
de Saram, G. S. W. & Pieris, M. V. P., 447 (Hel.)
Sarkar, N. K. & Maitra, S. R., (846) (Vms.)
——, —— & Roy, P. K., (846) (Vms.)
Sarrouy, Combe & Claude, 55 (Leish.)
Sarrouy, C., Cabannes & Roche, 807 (Leish.)
—— & Combe, P., (319) (Am.)
Sashin, D., Brown, G. N., Laffer, N. C. & McDowell, H. C., with Boeing, P. J., 460 (Der.)
Sasse, B. E., with Harper & Lisansky, 953 (Mal.)
Sati, M. H., with Kirk, 885 (Leish.)
Saunders, G. M., Bianco, A. A. & Jordan, W. S., 108 (Hel.)
——, with ——, Levine & Cohn, 1034 (Mal.)
Sautet, J. & Audibert, Y., 131 (Ent.)
Saxe, L. H., Jr., with Hauschka & Blair, 1054 (Tryp.)
Sayen, J. J., Pond, H. S., Forrester, J. S. & Wood, F. C., 305 (Typh.)
Scandar Bey, R., 1010 (Hel.)
Schäfer, W. & Reuter, A., 1061 (Typh.)
Scharff, J. W., 795 (Mal.)
Schein, J., with Adlersberg, 1087 (Sp.)
Schena, A. T., with Brachetto-Brian, 1082 (Hel.)
Schensnovich, V. B & Smirnova, E N., 1000 (Am.)
Schiavi, A., 165 (Mal.)
Schilling, I., with Kikuth, 199 (Typh.)
Schireson, R., with Kessel, Moore & Kaplan, 524 (Am.)
Schmidt, V., 231 (Def. Dis.)
Schneider, D. J., with Markell & Mullinger, 597 (Hel.)
Schneider, L. A. & Shallenberger, P. L., 1033 (Mal.)
Schnitzer, R. J., Lafferty, L. C. & Buck, M., 185 (Tryp.)
Schoch, E. W., with Gingrich, 970 (Mal.)
——, with ——, Schwab & Shepherd, 803 (Mal.)
Schroeder, E. F., with Most, Kane, Lavietes, London & Hayman, 496 (Mal.)
Schroeder, H. O., with Madden, Knipling & Lindquist, 544 (Ent.)
——, with —— & Lindquist, 1042 (Mal.)
Schueler, F. W., Chen, G. & Geiling, E. M. K., 1052 (Tryp.)
Schüffner, W. A. P., with Swellengrebel, 448 (Hel.)
Schwab, M., with Gingrich, Schoch & Shepherd, 803 (Mal.)
Schwachman, H., with Rodriguez-Molina, 829 (Hel.)
Schwetz, J., 508 (Tryp.), 786 (Mal.), 822 (R.F.), 1009 (Hel.)
—— & Dartevelle, E., 825 *bis* (Hel.)
Science To-Day, 942 (B.R.)
Scott, A. V., (1092) (Haem.)
Scott, E. G., with Flinn, Howard & Todd, 413 (Typh.)
Scott, J. A., 447, 839 (Hel.)
—— & Cross, J. B., (447) (Hel.)
——, Stembridge, V. A. & Sisley, N. M., 840 (Hel.)
Seager, L. D., with Richardson, Hewitt, Brooke, Martin & Maddux, 289 (Mal.)
Seal, S. C., 208 (Chl.)
——, with Lal, 144 (Reports, etc.)
Seaton, D. R. & Stoker, M. G. P., 576 (Typh.)
Sebah, P., with Benhamou & Nouchy, 238 (Misc. Dis.)
See, J. K., with Faust & Ingalls, 331 (Hel.)
Seeler, A. O. &Malanga, C., (399) (Mal.)
—— & Ott, W. H., 41, 185 (Mal.)
Segretain, G., (849) (Der.)
Seijas Rivas, J. V., 297 (Leish.)
Seitz, E., 829 (Hel.)
Selby, C. H., 398 (Mal.)
Selimkhanov, K. A., 91 (Am.)
Sellick, G., with Etherington, 19 (Mal.)
——, G., with ——, Hickling & Roden, 163 (Mal.)
Sen, N. R., with Dharmendra, 324, (430) (Lep.)
Sen, S. N., 209 (Chl.)
——, Basu, P. N. & Chakraborty, D. C., 209 (Chl.)
Sen Gupta, A. C., (588) (Ys.)
Sen Gupta, G. P., with Bhattacharji & Singh, 546 (Lab.)
Sen Gupta, P. C., 652, (1055) (Leish.)
—— & Chakravarty, N. K., (807) (Leish.)
——, with Dharmendra & Bose, 189 (Leish)
Senevet, G., (1020) (Ent.)
—— & Abonnenc, E., (131) (Ent.)
Sergent, Ed. & Sergent, Et., 36, 42, (Mal.)
——, —— & Collignon, E., 692 (Mal.)
Sergent, Et., 534, 535 *bis* (846) (Vms.)
——, with Sergent, Ed., 36, 42 (Mal.)
——, with —— & Collignon, 692 (Mal.)
Sergijev, P. G., 629 (Reports, etc.)
Serra, G., (510) (Tryp.)
Sewell, S. A., 347 (Der.)
Seydian, B., with Baltazard Mofidi & Bahmanyar, 906 (R.F.)

Shaffer, L. J. O., (679) (Misc. Dis.)
Shaffer, L. W. & Zackheim, H. S., (1093) (Der.)
Shaker, M., with Halawani, Nor-el-Din & Khalek, 99 (Hel.)
Shallenberger, P. L., with Lutterloh, 177 (Mal.)
——, P. L., with Schneider, 1033 (Mal.)
Shamanna, D. & Hedge, K. V., 520 (Pl.)
Shapland, C. D., 611 (Oph.)
Sharma, G. K. & Hussain, A., 737 (Hel.)
Sharma, S. N., with Basu, (860) *bis* (Reports, etc.)
Sharp, W. B. & John, M. B., 349 (Der.)
Sharpe, D. S., 463 (Misc. Dis.)
Shaul, J. F. & Saferstein, T. H., (665) (R.F.)
Shaw, E., with Bernstein, Stearns & Lott, (964) (Mal.)
Shaw, H. M., (223) (Hel.)
Sheiban, A., with Haddad & Budeir, 320 (R.F.)
Shen, S. C., Fleming, E. M. & Castle, W. B., 1045 (Mal.)
Shepard, C. C. & Topping, N. H., 573 (Typh.)
——, with —— & Huebner, 70 (Typh.)
——, with —— & Irons, 710 (Typh.)
Shepherd, C., with Gingrich, Schoch & Schwab, 803 (Mal.)
Sheppard, R. R., with Morgan & Barnett, 1039 (Mal.)
Shih Lu Chang, 83 (Am.), 665 (R.F.), 1055 (Leish.)
—— & Negherbon, W. O., 1056 (Leish.)
Shircore, J. O., 1017 (Vms.)
Shookhoff, H. B., Birnkrant, W. B. & Greenberg, M., 603 (Hel.)
Shortt, H. E., (511) (Leish.)
Shrager, J. & Kean, B. H., 170 (Mal.)
Shute, D., 521 (Am.)
Shute, P. G., 18, 169, 394 (Mal.)
Shwachman, H., with Bercovitz, 338 (Hel.)
Sicault, G., 426 (R.F.)
Sicé, A., 858 (Reports, etc.)
Siddons, L. B., 693 (Mal.)
Siedek, H., with Kiebl, 960 (Mal.)
Sierra Leone, 640 (Mal.)
Sievers, O., 890 (Typh.)
Siguier, F., with Blanc. (522), 524 (Am.)
Silva, M. S., 125, 538 (Der.)
da Silveira, M. M., with Kartman, 39 (Mal.)
Silverman, D. N. & Leslie, A., 819 (Am.)
Simeons, A. T. W. Chhatre, K. D., 77 (Pl.)
Simmons, J. S., (630 *bis*) (Reports, etc.)
Simmons, S. W., (182) (Mal.)
——, with Arnold & Fawcett, 277 (Mal.)
——, with Clapp & Fay, 563 (Mal.)
——, with Fay & Clapp, 562 (Mal.)
——, with Mathis & Ferguson, 644 (Mal.)
Simões, J. M. P., with Cambournac & Queiroz, 181 (Mal.)
Simons, H. C. R., 628 (Lab.)
Simons, R. D. G. P., 231 (Def. Dis.), 346, 535 (Der.)
Simpson, J. C. E., with Keneford, (1037) (Mal.)
Simpson, T., 588 (Ys.)
Simpson, W. F., with von Brand, (669) (Hel.)
——, with ——, Rees & Reardon, 84 (Am.)
Singer, R., 243 (Lab.)
Singh, G., (209) (Am.)
Singh, H., 623 (Misc. Dis.)
——, with Pringle, 853 (Ent.)
Singh, J., with Bhattacharji & Sen Gupta, 546 (Lab.)
Sinha, H. S., 86 (Am.)
Sinton, J. A., 282, 390 (Mal.)
Sirtori, C. & Zorzoli, G., 85 (Am.)
Sisley, N. M., with Scott & Stembridge, 840 (Hel.)
Skadovskaja, N. S., with Levinson, 888 (Leish.)
Sladden, R. A., with Maegraith, Brundrett & Rigby, 886 (Leish.)
Slavin, H. B., with Syverton, 623 (Prot.)
Smadel, J. E., Jackson, E. B., Bennett, B. L. & Rights, F. L., 513 (Typh.)
——, with Plotz, Bennett, Reagan & Snyder, 204 (Typh.)
——, with Robbins, Rustigian & Snyder, 64 (Typh.)
Smirnova, E. N., with Schensnovich, 100 (Am.)
Smith, C. E., Beard, R. R., Rosenberger, H. G. & Whiting, E. G., 537 (Der.)
——, ——, Whiting, E. G. & Rosenberger, H. G., 675 (Der.)
Smith, C. S., with Knowles, 399 (Mal.)
Smith, D. A., 604 (Def. Dis.)
Smith, F. D., with Mider & Bray, 610 (Der.)
Smith, F. H., (528) (Ys.)
Smith, P. K., 792 (Mal.)
——, Gallup, B. N. & Cain, L. J., 177 (Mal.)
Smithburn, K. C. & Haddow, A. J., 71 (Y.F.)
——, with ——, Mahaffy & Bugher, 992 (Y.F.)
——, with Mahaffy & Hughes, 72 (Y.F.)
Smolenskaja, J. M., 875 (Mal.)
Smyth, E. A., 237 (Ulc.)
Smyth, J. D., (919) (Hel.)
Snijders, E. P., Polak, M. F. & Hoekstra, J., 993 (Y.F.)
Snodgrass, R. E., (626) (Ent.)
Snyder, J. C., (989) (Typh.)
——, with Morgan & Stevens, 808 (Typh.)
Snyder, M. J., with Plotz, Smadel, Bennett & Reagan, 204 (Typh.)
——, with Robbins, Rustigian & Smadel, 64 (Typh.)
Snyder, T. L. & Meleney, H. E., 582 (Am.)
Soeiro, A. N., (44) (Bl.)
Sofia, F., with Ciaravino, 495 (Mal.)
Sofiev, M. S. & Leitman, M. Z., 906 (R.F.)
Sofiyev, M. S., 875 (Mal.)
——, with Lisova & Eskin, 876 (Mal.)
Sokhey, S. S., with Hazra & Lahiri, (235) (Vms.)
—— & Wagle, P. M., 418 (Pl.)
Soliman, H. S. & Mohamed, A. H., 458 (Vms.)
van Someren, E. C. C., (626), 680 (Ent.)
Sommer, H., with Baker, Foster, Meyer & Meyer, 712 (Pl.)
Sommers, A. H., with Campbell, K. N. Kerwin & Campbell, B. K., (34) (Mal.)
Soong, T. H., with Ho & Hsu, 987 (Leish.)
Soper, F. L., Davis, W. A., Markham, F. S. & Riehl, L. A., 892 (Typh.)
——, Knipe, F. W., Casini, G., Riehl, L. A. & Rubino, A., 877 (Mal.)
Soriano Lleras, A., 1029 (Rab.)
Sorsby, A. & Ungar, J., 616 (Oph.)

Sotolongo, F., with Basnuevo, 585, (663) (Am.)
Soubigou, X., (205) (Den.), (238), (Misc. Dis.)
——, Autheman & Guennec, 585 (Am.)
——, with Quérangal des Essarts & Autheman, (664) (Am.)
South Africa, Union of, 138, 856 (Reports, etc.)
Southern Rhodesia, 140 (Reports, etc.)
de Souza-Araujo, H. C., (719), 1072 (Lep.), 760 (B.R.)
Spensley, P. C., with Acheson & King, 964 (Mal.)
Sphangos, J. C., with Livadas, 259 (B.R.)
Spies, T. D., 609 (Haem.)
——, with Augulo, 1016 (Sp.)
——, with Frommeyer, 746 (Haem.)
——, with Lopez, Menendez & Toca, 456 (Sp.)
——, Lopez, G. G., Milanes, F. & Aramburu, T., 845 (Sp.)
——, with Milanes, Curbelo, Rodriguez & Kouri, 452 (Sp.)
—— & Stone, R. E., 533 (Haem.)
——, with Suárez & Suárez, 1089 (Sp.)
——, with Vilter & Vilter, 747 (Haem.)
Spinks, A., 35, 793 (Mal.)
——, with Butler & Davey, 1037 (Mal.)
—— & Tottey, M. M., 34 (Mal.)
Spitz, S., 1032 (Mal.)
Sprague, H. B., 25 (Mal.)
Stage, H. H., (181) (Mal.)
Stamps, P., with Huebner & Armstrong, 411 (Typh.)
Stannus, H. S., 282 (Mal.), 527 (Ys.), 683 (Reports, etc.)
Stare, F. J., with Roos & Hedgsted, 403 (Mal.)
Starkey, G. S., with Nelson, Webb & Bayliss, 229 (Hel.)
Stauber, L. A. & Walker, H. A., 967, 1046 (Mal.)
Stearns, B., with Bernstein, Dexter & Lott, (964) (Mal.)
——, with ——, Shaw & Lott, (964) (Mal.)
Stein, A. A. & Dorofejew, W. N., (214) (Lep.)
Steinberg, A., with Gall, 876 (Mal.)
Steiner, F., 891 (Typh.)
Steinhauer, L., 987 (Leish.)
Steinitz, H., 307 (Typh.)
Stembridge, V. A., with Scott & Sisley, 840 (Hel.)
Stening, S. E. L., 353 (Misc.)
Stephens, P. A. & Pratt, H. D., 564 (Mal.)
Stephenson, R. W., 598 (Hel.)
Stevens, A. L. B. & Blackman, G. G. B., 169 (Mal.)
Stevens, D. A., with Morgan & Snyder, 808 (Typh.)
Steward, J. S., (242), 855 (Ent.)
Stewart, G. T., 818 (Am.)
Stewart, M. O., with Black & Florio, 899 (Den.)
——, with Florio, (711) (Den.)
Stirewalt, M. A., with Kuntz, 434 (Hel.)
Stock, F. E., 136 (Ent.)
Stoker, M. G. P., with Seaton, 576 (Typh.)
Stoll, N. R., 667 (Hel.)
——, Chenoweth, B. M., Jr. & Peck, J. L., Jr., 737 (Hel.)
——, with Loughlin, 335, 735 (Hel.)
Hall, N. R., with Laughlin, Harris & Chenoweth, Jr., 444 (Hel.)
Stone, R. E., with Spies, 533 (Haem.)
Stowman, K., 208, 421 (Chl.)
Stransky, E. & Regala, A. C., 343 (Haem.)
Stuart, G., 193 (Typh.)
Stuart-Harris, C. H., with van den Ende, Fulton, Niven, Andrewes, Begg, Elford, White, Hawley, Mills, Hamilton & Thomas, 472 (B.R.)
Stubbs, T. H., with Young & Ellis, 492 (Mal.)
——, with ——, ——, Burgess & Eyles, 275 (Mal.)
Stubenbord, W. D., 528 (Ys.)
Stuczynski, L. A., with Bernkopf & Olitzki, 587 (R.F.)
Studdert, T. C., (1034) (Mal.)
Stunkard, H. W., (96), 432 (Hel.)
Suarez, J., (597) (Lep.), (735) (Hel.)
Suárez, J., (597) (Lep.)
Suárez, R. M., with Hernández Morales, Pratt & Oliver Gonzalez, 218 (Hel.)
——, Spies, T. D. & Suárez, R. M., Jr., 1089 (Sp.)
——, Welch, A. D., Heinle, R. W., Suárez, R. M., Jr. & Nelson, E. M., 607 (Sp.)
Suárez, R. M., Jr., with Suárez & Spies, 1089 (Sp.)
——, with ——, Welch, Heinle & Nelson, 607 (Sp.)
Sullivan, C. J. & Broun, G. O., 532 (Haem.)
Sullivan, R. R. & Ferguson, M. S., 435 (Hel.)
Sumerford, W. T., with Odum, (467) (Ent.)
Sundaresan, B., with Rao & Rao, 952 (Mal.)
Susini, R., with Manceaux, (239) (Misc. Dis.)
——, with —— & Bardenat, 849 (Misc. Dis.)
Swanson, E. R., with Bazemore, Johnson & Hayman, 559 (Mal.)
Swaroop, S., with Yacob, 157 (Mal.)
Swartzwelder, J. C., 225 (Hel.)
Sweets, H. H., Jr., with Packchanian (651) (Tryp.)
Sweigert, C. F., Turner, J. W. & Gillespie, J. B., 460 (Der.)
Swellengrebel, N. H., 285 (Mal.)
—— & Kraan, H., 286 (Mal.)
—— & Schüffner, W. A. P., 448 (Hel.)
Swendseid, M. E., with Bethell, Meyers, Andrews, Bird & Brown, (533) (Haem.)
——, Bird, O. D., Brown, R. A. & Bethell, F. H., (533) (Haem.)
Swyngedauw, J., Le Bourdellès, B. & Masse, L., (789) (Mal.)
Symes, C. B., 367, 853 (Ent.)
—— & Hadaway, A. B., 545 (Ent.)
Syverton, J. T. & Slavin, H. B., 623 (Prot.)

T.

Taggart, J. V., with Brodie & Udenfriend, 793 (Mal.)
Taillefer-Grimaldi, J., with Floch & de Lajudie, 93 (R.F.)
Takos, M., with Elmendorf & Barnhill, 882 (Mal.)
Takos, M. J., (1099) (Prot.)
Taliaferro, L. G., with Taliaferro. W. H., 965 (Mal.)
——, with ——, 40 (Mal.)

Taliaferro, W. H., 464 (Prot.), 493 (Mal.)
—— & Taliaferro, L. G., 40, 965 (Mal.)
Talice, R. V., 1076 (Hel.)
Tallant, E. J. & Maisel, A. L., with Rakestraw, M. R., 82 (Am.)
Tanguy, Y., with Chorine, 211 (Am.)
Tarzwell, C. M., (755) (Ent.)
—— & Fisk, F. W., 563 (Mal.)
Taylor, C. E., with Kean, 166 (Mal.)
Taylor, F. H., 476 (B.R.)
Taylor, R. M. & da Cunha, J. F., 414 (Y.F.)
——, with Laemmert & Ferreira, 414 (Y.F.)
——, with Waddell, 309 (Y.F.)
Taylor, S. E., 618 (Heat Str.)
Tchernomoretz, I., with Adler, 406 (Leish.)
Teasley, G. H., 172 (Mal.)
Teixeira, A. W. G., with de Azvedo, 981 (Leish.)
Templeton, W. C., 896 (Typh.)
Teodorescu, A. M., with Duport, 996 (Den.)
——, with Zotta, Radacovici, Cantacuzène & Lupasco, 623 (Misc. Dis.)
Terzian, L. A., 969 (Mal.)
Tesar, W. C., with Cox & Irons, 710 (Typh.)
Theodor, O., 853 (Ent.)
Theron, P., 1002 (Am.)
Thetford, N. D., with Brown, 338 (Hel.)
Thibault, C., with Thuret, (666) (Hel.)
van Thiel, P. H., 787 (Mal.)
—— & Weurman, C., 557, 787 (Mal.)
Thomas, C. C., with van den Ende, Stuart-Harris, Fulton, Niven, Andrewes, Begg, Elford, White, Hawley, Mills & Hamilton, 472 (B.R.)
Thomen, L. F. & Read, H., 210 (Am.)
Thompson, C. M. & White, B. V., Jr., 79 (Dys.)
Thompson, P. E., 43, 569 (Mal.)
——, with Burks, 124 (Der.)
Thompson, W. E. F., 803 (Tryp.)
Thomson, A. M. & Freedman, B., 673 (Def. Dis.)
Thomson, R. C. Muirhead, 795 (Mal.)
Thorne, B. T., 615 (Oph.)
Thornell, H. E., with Henderson, 609 (Haem.)
Thuret, C. & Thibault, C., (666) (Hel.)
Tiant, F. R., with Pardo-Castello & Piñeyro, 1074 (Lep.)
Tibbs, R. O., with Diercks, 894 (Typh.)
Tiburskaya, N. A., with Pospelova-Strom, 213 (R.F.)
Tillinghast, A. J., with Weinberg, 441 (Hel.)
Tillman, A. J. B., 600 (Hel.)
Timpano, P., 299 (Leish.)
Tisseuil, J., (214), (215), 325, 726 (Lep.), 618 (Oph.)
Titus, H. A., with Jones & Oberg, (467) (Ent.)
Tobback, L., 259 (B.R.)
Toca, R. L., with Lopez, Spies & Menendez, 456 (Sp.)
Toce, A., with Bacigalupo & Rey, 529 (Hel.)
Todd, A. R., with Hull, Lovell & Openshaw, (642) (Mal.)
Todd, C. W., with Flinn, Howard & Scott, 413 (Typh.)
Toffaleti, J. P. & King, W. V., 958 (Mal.)
Tomich, P. Q., 996 (Pl.)
Tomlinson, W. J., with Einhorn, 393 (Mal.)
Tonkin, I. M., 184 (Mal.)
Topping, N. H., with Shepard, 573 (Typh.)
——, —— & Huebner, R. J., 70 (Typh.)
——, —— & Irons, J. V., 710 (Typh.)
Torode, K. R., 278 (Mal.)
Torrance, H. W. (835) (Hel.)
Torrealba, J. F., 187 (Tryp.)
Tottey, M. M., with Maegraith, Adams, Andrews & King, 501 (Mal.)
——, with Spinks, 34 (Mal.)
Toulant, P. & Larmande, A., 1095 (Oph.)
Tovarnickij, V. I., Krontovskaja, M. K. & Ceburkina, N. V., (301) (Typh.)
Tow, L. R., with Gunther, 133 (Ent.)
Towbin, E. J., with Molnar, Gosselin, Brown & Adolph, 461 (Heat Str.)
Towson, R. T., with Ryder, 393 (Mal.)
Trager, W., 1046 (Mal.)
——, Bang, F. B. & Hairston, N. G., 1036 (Mal.)
——, with ——, —— & Maier, 281, 503, 696 (Mal.)
Trapido, H., 287 (Mal.)
Traquair, R. N., 751 (Misc. Dis.)
Travis, B. V., (839) (Hel.)
——, Maple, J. D., Hurlbut, H. S. & Husman, C. N., 646 (Mal.)
Tredre, R. F., 488, 795 (Mal.)
Trembley, H. L., 402 (Mal.)
——, with Josephson & Culwell, 1030 (Mal.)
Trimarchi, E., 168 (Mal.)
Trincão, C., (1092) (Haem.)
Trinquier, E., with Ceccaldi, Paquier, Pelissier & Vargues, 949 (Rab.)
Tropical Medicine News, 248 (Reports, etc.)
Trupp, M., 495 (Mal.)
Trussell, R. E., 679 (Prot.)
Tshinaev, P. P., 680 (Ent.)
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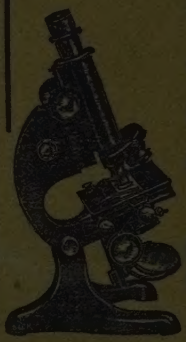

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